

The background of the cover features a detailed anatomical illustration. At the top, a human brain is shown in a frontal view. Below it, a heart is depicted with its major blood vessels (aorta, pulmonary artery, and pulmonary veins) and coronary arteries. A blue ECG (heart rate) line is drawn across the heart and extends towards the left edge of the cover. The entire illustration is set against a dark blue background with a subtle grid pattern.

ECG Pocket Brain

2014

**Expanded
Version**

e-Pub

Ken Grauer, M.D.

**6th Edition
(2014)**



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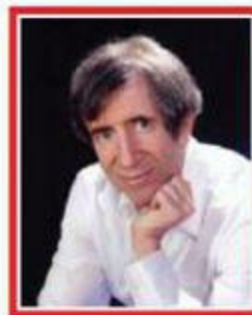
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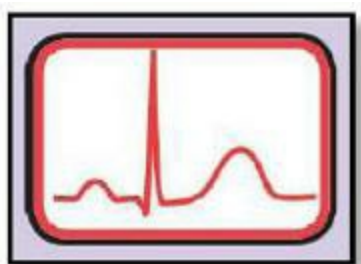
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— Acknowledgements / Copyright —

Sole Proprietor — Ken Grauer, MD

Design of All Figures — Ken Grauer, MD

Printing — by Renaissance Printing (*Gainesville, Florida*)

- *Special Acknowledgement to Colleen Kay (for making the hard copy version of this book happen) and to Jay (for all things technical).*

Special Dedication:

- To Cathy Duncan (*who is my wife, my best friend, and the LOVE of My Life*).

Additional Acknowledgements:

- Rick & Stephanie of Ivey's Restaurant (*great food, staff and atmosphere that inspired my ACLS creativity*).
- Abbas, Jane, Jenny & Gerald of the Haile Village Bistro (*for great food at my other writing space*).



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- 6th Edition (*2014*) plus ePub-2014 edition.

written consent from the publisher.

- **ISBN # 978-1-930553-30-9** (# *1-930553-30-7*)

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– About ECG-2014-ePub

Electrocardiography is *not* difficult. At least it is not difficult to obtain a basic understanding of the art — and *apply* this understanding to interpreting the *majority* of 12-lead ECGs and arrhythmias that you will encounter. The most difficult part of electrocardiography is learning (*and then remembering*) the various criteria used to diagnose complex ECG findings such as chamber enlargement and bundle branch block.

- Practically speaking — ***there is much less to learn (and memorize) than most people think.*** Herein lies the *secret* of the ECG Pocket Brain: it facilitates understanding of basic ECG concepts and *lightens* the "memory load" — by providing *ready* recall of those *KEY* facts and figures needed for successful 12-lead interpretation.
- We have ***completely revised and updated*** this **6th Edition** (2014) of our book. We have more than doubled our content — enhanced our explanations — and have greatly improved the quality of our figures. This book now stands as an *independent* concise text on key aspects of ECG interpretation.
- ***Development of this ePub*** takes ECG-2014-PB (*Expanded*) to a *higher* level. Nothing beats the instant access of a well organized electronic file.



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- For readers who are *less* experienced in *systematic* ECG interpretation — We suggest you refer to the ECG Crib Sheet often. Doing so will facilitate ready recall of the 6 *KEY* parameters (*of Rate-Rhythm-Intervals-Axis-Hypertrophy-QRST Changes*).
- **HINT:** Making a bookmark — ***Searching*** for, “00.6” — or simply *scrolling* to the front of this ePub are *easy* ways to rapidly access the ECG Crib Sheet.

- *Please CHECK OUT our ECG Crib Sheet!*

NOTE-2: We have placed review of the **6 Essential Lists** in **Section 00.7**. This also appears at the front of this ePub (*before Section 01.0*) for ready access.

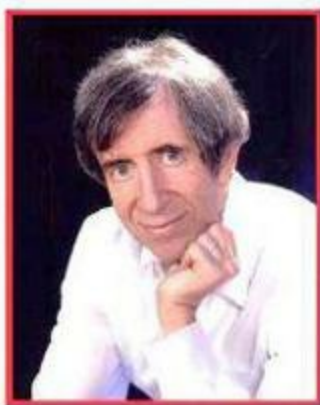
- You will want to refer to these 6 Lists often until you know them by heart. With *minimal* practice — they will be *easy* to remember!
- **HINT:** Making a bookmark — **Searching** for, “**00.7**” — or simply *scrolling* toward the front of this ePub (*just after the ECG Crib Sheet*) are *easy* ways to rapidly access the 6 Essential Lists.

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– About the Author –



● **Ken Grauer, MD** has taught ECG interpretation for more than 3 decades. Author of more than 10 books (*which have sold half a million copies*) – he is a family physician educator whose medical passion is cardiology. His other “passions” are foreign languages (*Fr./Sp./Ger.*) — as well as dancing (*ballroom/2-step/Argentine tango*).

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00.6.1 – The ECG Crib Sheet: *How BEST to Use It*

This ePub book begins in Section 01.0 with *Review of Basics*. We *intentionally* place this **ECG Crib Sheet** before Section 01.0 — so that it will be *easy* to access.

- For readers who are *less* experienced in *systematic* ECG interpretation — We suggest you refer **often** to this ECG Crib Sheet. This will **facilitate ready recall** of the 6 *KEY* parameters in the **Systematic Approach** (= *Rate-Rhythm-Intervals-Axis-Hypertrophy-QRST Changes*).
- With *regular* use — You'll *quickly* learn the information contained within this ECG Crib Sheet. In case there is something you forget — the Crib Sheet is always here for a quick refresher.
- **HINT:** Making a bookmark — **Searching** for, “00.6” — or simply *scrolling* to the front of this ePub are *easy* ways to rapidly access the ECG Crib Sheet.
- **NOTE:** *All* parameters in our *Systematic Approach* to ECG interpretation are discussed in detail throughout the pages of this ePub. We begin with **Section 01.0** on *Review of Basics* — and continue through to Section 14.0. *We simply want you to know* that this ECG Crib Sheet (*Section 00.6*) and Section 00.7 that follows (*which reviews the 6 Essential Lists*) — are **located** at the very *front* of this **ePub** in order to facilitate your *ready* access to this material.

00.6.2 – The *Systematic Approach*: *Rate & Rhythm*

- The first 2 parameters in the **Systematic Approach** are Rate and Rhythm. They are discussed in detail in **Section 02.0**.

RATE — If the rhythm is regular — **rate** can easily be determined by the “Rule of 300”. Divide 300 by the number of boxes in R-R Interval (*Section 02.2*).

- IF the rhythm is **regular** and **rapid** — rate can be accurately estimated by the *every-other-beat* method (*Section 02.31*) — in which you figure out the rate for every-other-beat (*which is half the rate*); and then *double* this number.

RHYTHM — First **ensure** that the patient is **hemodynamically stable**. Then assess the 5 *KEY* components for rhythm. These are conveniently remembered by the phrase: **Watch your P's & Q's — and the 3 R's** (*Section 02.1*).

- Are there **P waves**? If so — Are P waves **upright** in lead **II**?
- P waves should *always* be **upright** in lead **II** IF there is sinus rhythm (*unless there is lead*

reversal or dextrocardia).

- Is the **QRS** complex wide or narrow?
- What is the **Rate**?
- Is the rhythm **Regular**?
- Are P waves **Related** (ie, "married" with fixed PR interval) to the QRS? If P waves are "married" — then they are being conducted to the ventricles.

00.6.3 – Parameter #3: Intervals

INTERVALS — Look at intervals at an *early* point in the process!

- The **PR Interval** — is *prolonged* IF $>0.20-0.21$ second (if *clearly more than a LARGE box in duration*) — Section 04.0.
- The **QRS Complex** — is *wide* IF >0.10 sec. (if *more than HALF a large box in duration*) — Section 05.0.
- The **QT Interval** — is *prolonged* IF *clearly more than half* the R-R interval (provided that heart rate is not more than 100 beats/minute) — Section 06.0. If the QT interval is prolonged — Think of "Drugs-Lytes-CNS" (= **List #3**) as the possible cause(s) of QT prolongation (Section 06.2).

KEY Clinical Point: — IF the rhythm is sinus but the QRS is wide — then **STOP before** going further. Figure out *WHY* the QRS is wide — be this due to RBBB, LBBB, IVCD, WPW (Section 05.2). Criteria for infarction, ischemia, and chamber enlargement will all be *different* IF there is a conduction defect. This is the reason for determining *early on* the cause of QRS widening.

00.6.4 – Parameter #4: Axis

- Axis is discussed in detail in **Section 07.0**.

AXIS — Determine the axis quadrant by looking at **lead I** (at 0 degrees) and **lead aVF** (at +90 degrees):

- The axis is **normal** — IF the *net* QRS deflection is *positive* in leads I and aVF (this defines the axis to be between 0 degrees to +90 degrees).
- There is **RAD** — IF the *net* QRS deflection is *negative* in I but *positive* in aVF (Think RVH, LPHB or normal variant as the common causes for RAD).
- There is **LAD** — IF the *net* QRS is *positive* in I but *negative* in aVF. There is *pathologic LAD* = **LAHB** (Left Anterior HemiBlock) — IF the *net* QRS deflection is more *negative* than positive in lead II (Section 07.13).
- The axis is **indeterminate** — IF the *net* QRS deflection is *negative* in leads I and aVF (Think RVH, COPD, obesity as the common causes of an indeterminate axis).

00.6.5 – Parameter #5: *Chamber Enlargement*

- Chamber Enlargement is discussed in detail in **Section 08.0**.

HYPERTROPHY (*Chamber Enlargement*):

- The "*magic numbers*" for **LVH** are **35** (sum of the *deepest* S wave in V1,V2 — *plus* — the *tallest* R wave in V5,V6 — *in a patient at least 35 years of age*) — and **12** (for R wave amplitude in lead aVL) — *Section 08.1*. True chamber enlargement is *much* more likely IF "*strain*" also present!
- There is **RAA** (*P Pulmonale*) — IF P waves are prominent (≥ 2.5 mm tall) and peaked (ie, "*uncomfortable to sit on*") in the *pulmonary* leads (*II, III, and aVF*) — *Section 08.19*.
- There is **LAA** (*P Mitrale*) — IF P waves are *notched* ("*m*"-shaped) in *mitral* leads (*I, II, or aVL*) — or if the P wave in V1 has a *deep terminal negative* component — *Section 08.20*.
- Consider **pulmonary disease** — IF there is RAA, RAD (*or indeterminate axis*), incomplete RBBB (*or rSr' pattern in lead V1*), low voltage, or persistent precordial S waves — *Section 08.24*.
- Consider **RVH** — IF there is *also* a tall R wave in V1 *and/or* right ventricular "*strain*" — *Section 08.24*.
- Criteria for LVH/RVH are *different* when there is BBB (*Sections 05.31 and 05.32*).
- Consider **acute pulmonary embolus** — IF the ECG shows signs of *acute right heart strain* (*Section 08.34*).

00.6.6 – Parameter #6: *Infarct* (= Q-R-S-T Changes)

- Assessment of QRST Changes is discussed in detail in **Section 09.0**.

INFARCT (= **Q-R-S-T changes**) — Look at *all* 12 leads on the ECG to assess for QRST Changes.

- **Lead aVR** will usually be **globally negative** under *normal* circumstances (*negative P wave, QRS and T wave*). We therefore *expect* to see a Q wave and T wave inversion in lead aVR as part of a normal ECG.
- The finding of an *upright* P, QRS and T wave in lead aVR should suggest the possibility of **lead misplacement** (*especially if lead I manifests global negativity*) — *Section 03.10*.
- For the *more* experienced interpreter — We suggest review of Sections 09.31-through-09.39, which highlight situations for which **attention** to **lead aVR** may provide important additional information.

NOTE: *Localization* of the **Basic Lead Groups** is shown in Figure 00.6.6-1. Lead localization is discussed in detail in *Section 09.4*.



Figure 00.6.6-1: Basic Lead Groups.

Assessment of Q-R-S-T Changes:

- **Q Waves** — Small and normal *septal q waves* are commonly seen in one or more of the *lateral* leads (I, aVL, V₄, V₅, V₆).
- **Moderate or large-sized Q waves** may be **normal** — IF they occur as an *isolated* finding in *one or more* of the following leads: leads III, aVF, aVL, and/or V₁. Figure 00.6.6-2 suggests that thinking of a **reverse Z** (as in Zorro) — may help recall *which* leads may *normally* manifest a large *isolated* Q wave (Section 09.12).

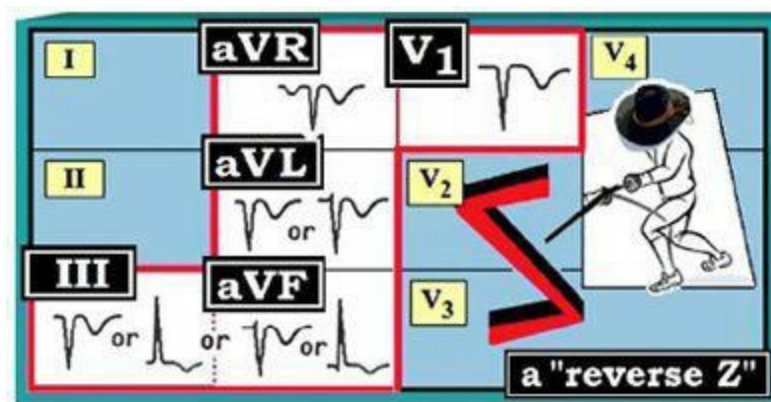


Figure 00.6.6-2: Reverse Z memory aid (Q waves/T inversion).

- **R Wave Progression** — Does *transition* occur as it normally should *between* leads V₂-to-V₄? (Figure 00.6.6-3 — Section 09.5).
- Is there a **Tall R in Lead V₁**? = **List #6** (Section 10.47).
- Normally the QRS is predominantly *negative* in *right-sided* lead V₁. Is there an **rSr'** pattern in **lead V₁**? (which could be a normal variant or incomplete RBBB) — Section 05.18.

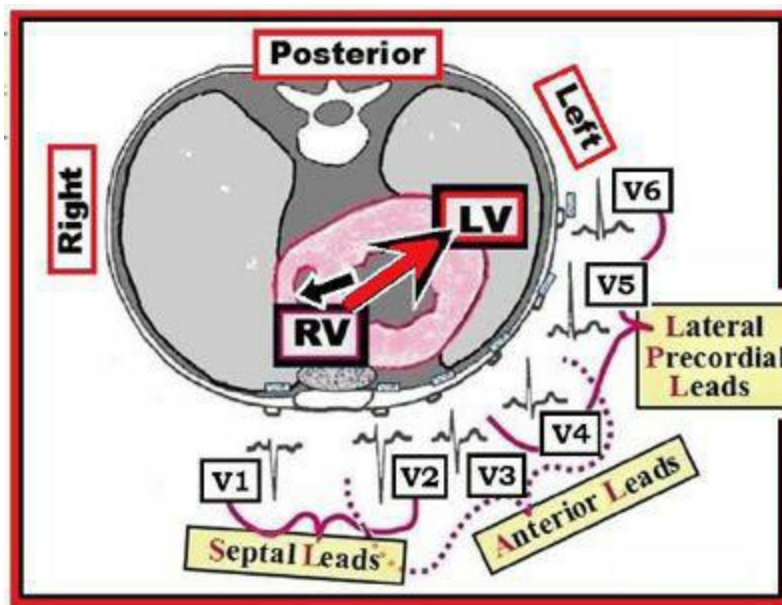


Figure 00.6.6-3: Precordial lead placement. Normal R wave progression.

- **ST Segments** — Judge ST segment deviation (*elevation or depression*) with respect to the PR segment baseline (*Section 09.25*). Much more than the *amount* of ST segment deviation — Focus on *shape* ("*smiley*" or "*frowny*").
- **T Waves** — May *normally* be inverted in leads **III**, **aVF**, **aVL** and/or **V1**. Note that these are the *same* leads that may normally manifest *isolated Q waves* (*Figure 00.6.6-2*).
- Remember that criteria for ischemia/infarction are *different* when there is BBB (*Sections 05.24-through-05.29*).

00.6.7 – Suggested Approach: Systematic ECG Interpretation

As we discuss in **Section 01.0** — Use the elements in *Sections 00.6.2 through 00.6.6* as a *guide* for **descriptive analysis**; then formulate your **clinical impression**. Whenever possible — *WRITE OUT* (or at least *think out*) your findings. Above all, even when time is short — *BE systematic!*



00.7.1 – The 6 Lists: *What They Are ...*

We have developed **6 Essential “Lists”** to remember for optimal ECG and arrhythmia interpretation. The purpose of a “List” — is that it readily recalls the most common/important causes to remember for the particular entity. For example — as soon as you recognize that the QT interval is prolonged — *Think “Drugs-Lytes-CNS” as possible causes (= LIST #3).*

- We intentionally *limit* the number (*and length*) of our lists so as *not* to overwhelm. Use of these 6 Lists will prove *invaluable* to you in *saving* time and improving the *accuracy* of your interpretation.
- You will want to refer to these 6 Lists often until you know them by heart. With *minimal* practice — they will be *easy* to remember!

For clarity (*and easy reference*) — we reproduce the 6 Lists here. You’ll find them again *intermingled* with relevant content *throughout* this electronic file.

- **HINT:** Making a bookmark — **Searching** for, “00.7” — or simply *scrolling* to the front of this ePub are *convenient* ways to instantly access the 6 Essential Lists (*The 6 Lists are found just after the ECG Crib Sheet*).

00.7.2 – LIST #1: The *Common Causes of a Regular WCT:*

- LIST #1 is discussed in detail in Section 02.47.
- **KEY Point:** The 1st thing to do in assessment of any tachycardia — is to **ensure hemodynamic stability**. IF the patient is unstable — *immediately* cardiovert! But IF the patient in WCT (Wide-Complex Tachycardia) is stable — you have at least a *moment* of time to contemplate management.
- Always **assume** a regular WCT rhythm is **VT until proven otherwise** (Figure 00.7.2-1). This is true regardless of whether the patient is hypotensive or alert with a systolic BP>160mmHg. Some patients may be stable and alert despite remaining in VT for hours (*or even days*)!
- The reason we list **VT** as the **first 8 causes** in **LIST #1** (Figure 00.7.2-1) is twofold: **i)** VT is the *worst* thing the WCT could be; and **ii)** Statistically — *at least* 80-90% of cases of WCT of *uncertain* etiology will turn out to be VT.

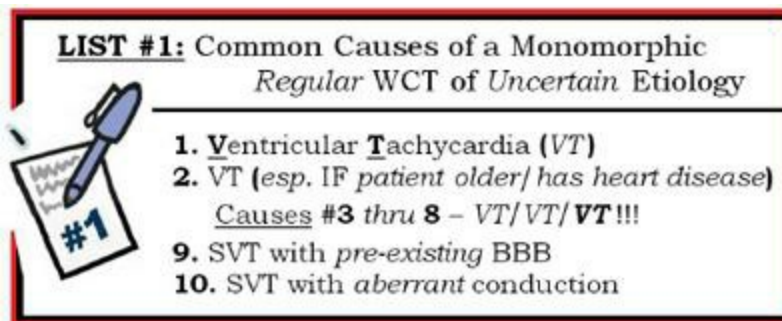


Figure 00.7.2-1: LIST #1 = Causes of a Monomorphic Regular WCT. Always assume VT until proven otherwise.

00.7.3 – LIST #2: Common Causes of a Regular SVT:

- LIST #2 is discussed in detail in Section 02.52.
- We define the term, “SVT” — as a *rapid* rhythm (*rate >100/minute*) in which the **QRS** complex is **narrow** (*not more than 0.10 second*) in *all 12 leads*. The **3 entities** included in **LIST #2** (Figure 00.7.3-1) make up over 90% of the causes of a **regular SVT** seen by primary care or emergency providers.

Awareness of the **usual rate ranges** for these 3 common causes of SVT may help with distinction between them:

- **Sinus Tachycardia** — *rarely* exceeds 160/minute in adults (*children may have sinus tachycardia at much faster rates*).
- **Untreated Atrial Flutter** — usually conducts with **2:1 ratio**. Since the atrial rate of *untreated* atrial flutter is almost always *close* to **300/minute** (*250-350/minute is the range*) — this means that the *ventricular* response to AFlutter will usually be **between 140-to-160/minute**.
- As a result — IF a *regular* SVT in an adult is *faster than* 160-170/minute — it is most likely to be PSVT (= AVNRT = AtrioVentricular Nodal Reentry Tachycardia).
- On the other hand — A *regular* SVT rhythm at a rate *under* 160-170/minute could be any of the 3 entities on LIST #2 (Fig. 00.7.3-1).
- **NOTE:** A **vagal maneuver** or use of Adenosine (= *chemical Valsalva*) — may help in making a definitive diagnosis.

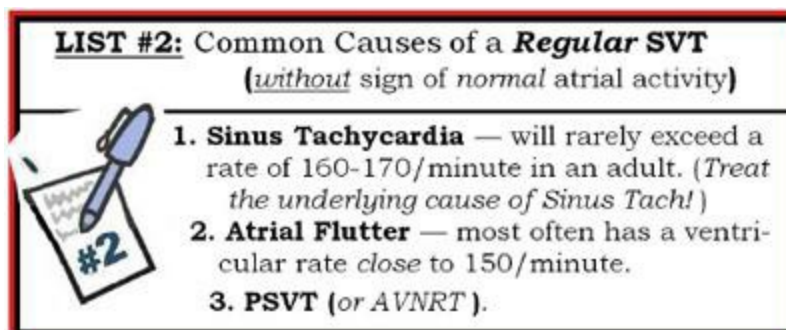


Figure 00.7.3-1: LIST #2 = Causes of a Regular SVT.

00.7.4 – LIST #3: Common Causes of QT Prolongation:

- LIST #3 is discussed in detail in Section 06.2.
- Be aware that *in addition* to the **3 entities** in **LIST #3** (Figure 00.7.4-1) — ischemia/MI/BBB may *all* lengthen the QT. That said — IF the *only* ECG abnormality on the tracing is a *long* QT — *Think “Drugs-Lytes-CNS”* as the likely cause. Then — *correlate* clinically.
- **NOTE:** Many *combinations* of **drugs** may contribute to *lengthening* the QT interval. These potential *drug-drug* interactions extend *beyond* the scope of this ECG-ePub and are *not* reflected in List #3. Suffice it to say that risk of QT prolongation (*and risk of Torsades*) is generally *minimal* — IF an otherwise *healthy* adult takes a *single* new drug for a *limited* period of time. Risk goes up when *several* agents are taken — especially if the drugs utilize the p450 system for metabolism and the patient has other *underlying* medical problems that may *predispose* to Torsades. **BOTTOM Line:** Consider the possibility of **Drugs** (*or drug-drug interactions*) — as one of the main clinical causes of QT prolongation.

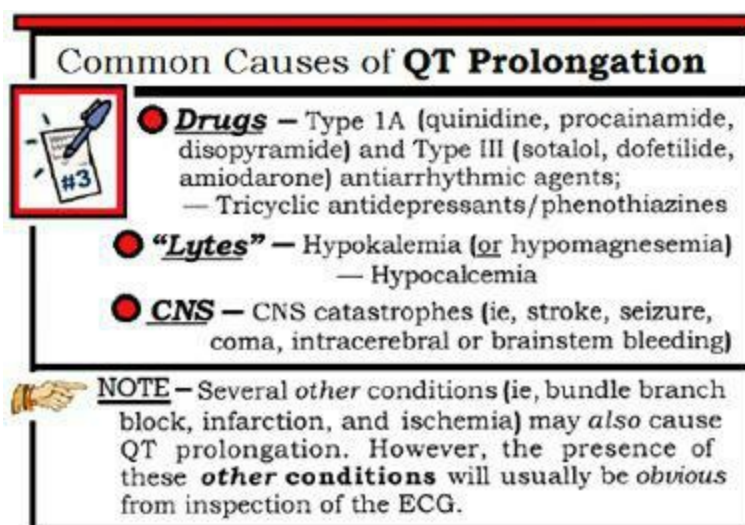


Figure 00.7.4-1: LIST #3 = Causes of a *Prolonged* QT.

00.7.5 – LIST #4: Common Causes of ST Segment Depression:

- LIST #4 is discussed in detail in Section 09.26.
- There are *over* 50 causes of ST depression. Many of these causes are noncardiac — related to factors such as hyperventilation, temperature extremes, severe medical illness. The entities in **LIST #4** (Figure 00.7.5-1) — are simply the ones we feel it most important to remember.
- A *similar* list of entities as shown in List #4 may produce T wave inversion.

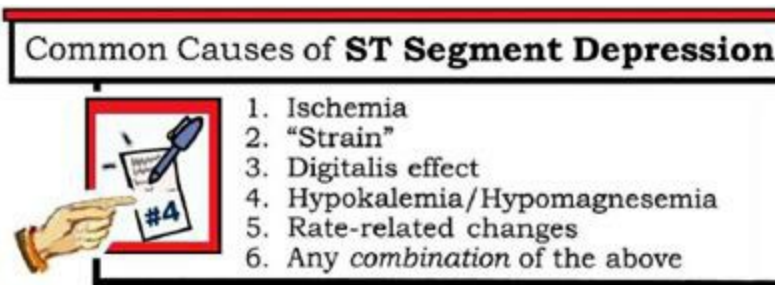


Figure 00.7.5-1: LIST #4 = Causes of ST Depression.

00.7.6 – LIST #5: Causes of *Anterior* ST Depression with *Inferior* MI:

- LIST #5 is discussed in detail in Section 10.42.
- Use of **LIST #5** (Figure 00.7.6-1) is *limited* to the setting of **acute inferior STEMI**. The purpose of List #5 is to remind us of the **3 common causes** of *anterior* ST depression in this situation.
- Helpful General Rule: The *more* ST depression that is seen on the ECG — the more likely the MI is both *acute* and *large*.
- Clinically — we may *not* be able to determine with certainty *which* of the 3 entities in List #5 is/are operative. *This does not matter!* What counts is *awareness* of a more *extensive* MI (*that is more likely to benefit from acute intervention*) — when significant *anterior* ST depression is seen in conjunction with acute *inferior* infarction.

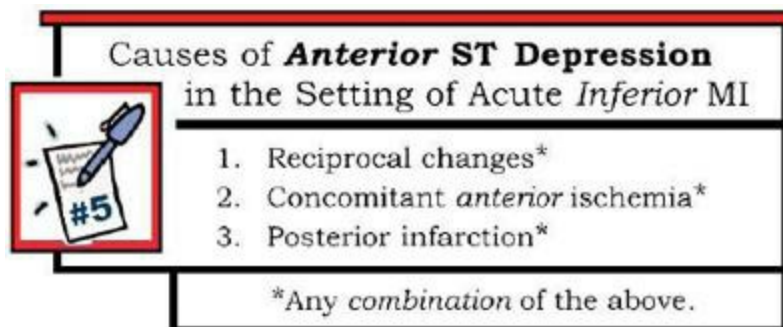


Figure 00.7.6-1: LIST #5 = Causes of *Anterior* ST Depression in the Setting of Acute *Inferior* MI.

00.7.7 – LIST #6: Common Causes of a *Tall* R in Lead V1:

- LIST #6 is discussed in detail in Section 10.47.
- Normally the QRS is predominantly *negative* (a *QS* or *rS* complex) in *right-sided* lead V1. As a result, the finding of even a *relatively* tall R wave in lead V1 (*that equals or exceeds S wave amplitude in this lead*) — should prompt consideration of the 6 entities on **LIST #6** (Figure 00.7.7-1).
- Determining *which* of the 6 entities in List #6 is present — is decided by assessing *associated* ECG findings. A normal variant (*Cause #6*) should be considered only after the first 5 causes have been *ruled out*.
- *Not* all patients need an Echo. On occasion, however — an Echo may be needed to *rule out* structural heart disease (*especially if hypertrophic cardiomyopathy is a consideration*).

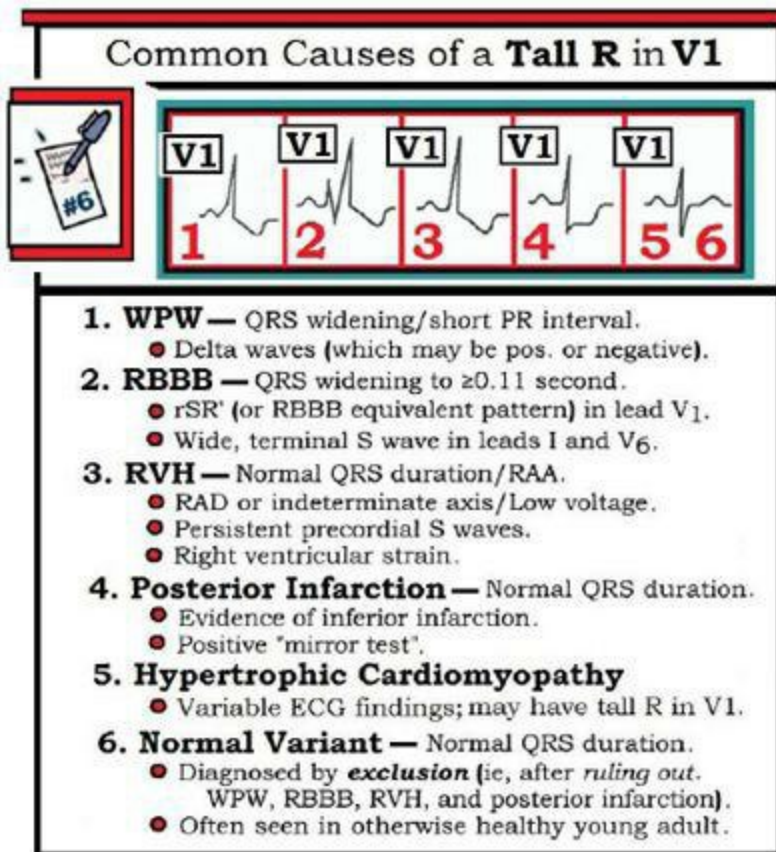


Figure 00.7.7-1: LIST #6 = Causes of a *Tall* R Wave in Lead V_1 .



Review of Basics

01.1 – Systematic Approach to 12-Lead ECG Interpretation

The **KEY** to interpretation of *any* ECG is to utilize a **Systematic Approach**. The approach we suggest for interpreting each 12-lead ECG you encounter entails sequential **systematic assessment** of the following parameters:

- **R**ate
- **R**hythm
- **I**ntervals (*PR/QRS/QT*)
- **A**xis
- **H**ypertrophy
- **I**nfarct (*QRST changes*)

We outline **KEY** elements to assess for each of the above parameters on our **ECG Crib Sheet** (See Section 00.6). Discussion here is limited to the following:

- The **purpose** of having (*and regularly using*) a *sequential* systematic approach is simple: It provides you with an *easy-to-remember* mental checklist that **prevents** you from **overlooking** potentially important findings.
- Additional benefits include increased **accuracy**, improved organization, and surprisingly — **increased speed** in completing your interpretation. With *regular* use — applying the system soon **becomes automatic!**

01.2 – The 2 Steps to Systematic Interpretation

The process of 12-lead ECG interpretation should be thought of as consisting of **two major steps**. The secret to successful interpretation depends on **keeping** these 2 steps **separate** in your mind:

- **Step #1** = Descriptive Analysis — is accomplished first: Simply *describe* what is seen on the tracing (*as per the checklist review on the ECG Crib Sheet in Section 00.6*). Ideally — **WRITE OUT** your findings (*or at least THINK out your findings if you do not have time to write them out*).
- **Step #2** = The Clinical Impression — should only come *after* Step #1 has been completed. Those *specific* findings identified in descriptive analysis should *now* be interpreted *in light of* the clinical context (*ie, as defined by the patient's age, presenting complaint, and additional relevant clinical history*).

01.3 – WHY 2 Separate Steps for Interpretation?

Review of the ECG shown in [Figure 01.3-1](#) provides insight as to why there is need for 2 separate steps to interpretation.

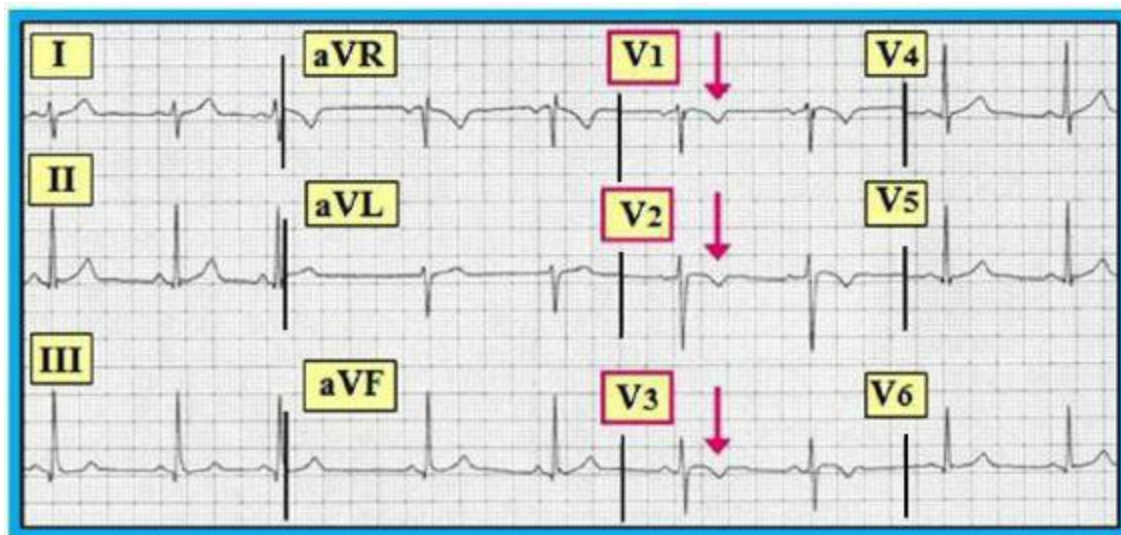


Figure 01.3-1: How would you interpret this 12-lead ECG?

Answer to Figure 01.3-1: We interpret the 12-lead ECG shown above by the 2-Step *Systematic* Approach:

Step #1 (Descriptive Analysis):

- **Rate and Rhythm** — Sinus arrhythmia at a rate of between 60-70 beats/minute.
- **Intervals (PR/QRS/QT)** — are normal.
- **Axis** — There is RAD (*Right Axis Deviation*) with a mean QRS axis of $\sim +100$ degrees.
- **Hypertrophy** — None.
- **Q-R-S-T changes** — Small Q waves in *inferolateral* leads; R wave progression is normal (*transition occurs between leads V3-4*); Assessment of ST-T wave changes reveals fairly deep **symmetric T wave inversion** in **anterior precordial leads** (arrows in [Fig. 01.3-1](#)).

Step #2 (Clinical Impression) — *It depends!* That is — How we would interpret the ECG in [Figure 01.3-1](#) will vary greatly depending on the **age and clinical history** of the patient.

- **Symmetric T wave inversion** as seen in the *anterior* leads (*V1, V2, V3*) of [Fig. 01.3-1](#) is common in pediatric patients. In an otherwise healthy child (*with no heart murmur*) — this finding represents a *benign* normal variant (referred to as a **Juvenile T Wave Pattern**).
- BUT — the *same* ECG (*with identical T wave inversion*) would have to be interpreted very differently IF the patient in question was an *older* adult with chest pain (*in which case ischemia would be suggested*). And, if *new-onset* dyspnea was the primary concern from the history — **acute pulmonary embolism** would also have to be considered ([Section 08.34](#)).
- **KEY Clinical Point: Descriptive Analysis** is the *same* in both cases (= *symmetric T wave inversion in V1-to-V3*) — but the *Clinical Impression* is very different!

Bottom Line: — Keep Steps #1 and 2 separate in your mind. Always do *Descriptive* Analysis first! Then — decide on your *Clinical* Impression of the findings identified in light of the clinical situation (*age of patient; clinical history*).



Rate & Rhythm

02.1 – Assessing the 5 Parameters of Rhythm

The most important clinical point (*and the real KEY to rhythm interpretation*) — is to utilize a **Systematic Approach**. The system we favor is based on assessing the rhythm for the following 5 parameters:

- **P** waves?
- **QRS** width ?
- **Regular** rhythm ?
- **Related** (*P waves related to QRS ?*)
- **Rate** (*heart rate ?*)

Memory Aid (*to remember the 5 Parameters*):

- "Watch your **P**'s and **Q**'s — and the 3 **R**'s" ...

NOTE: It does *not* matter in what *sequence* you look at these 5 parameters for rhythm — as long as you *always* check *each* of them *every* time you look at an ECG or rhythm strip! We often begin with that parameter that is *easiest* to assess (*be this presence of P waves, QRS width or regularity*).

- First — Ensure that the **patient** is **hemodynamically stable** (*precise rhythm diagnosis is of secondary importance if the patient is acutely decompensating and about to code*). **Always look at the patient first** — before beginning to study the rhythm.
- IF there is normal **sinus rhythm** — then **P** waves should be present and clearly **upright** in lead **II** (*See Section 02.3*).
- In adults, the **QRS** is defined as **narrow** — IF it is **≤0.10 second** = not more than **HALF** a large box in duration (**Figure 02.1-1**).

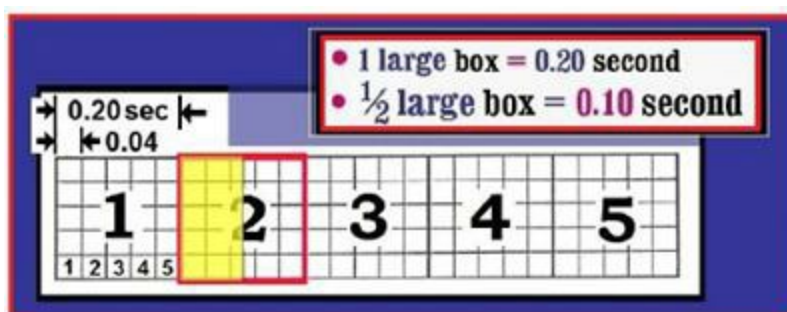


Figure 02.1-1: Is the QRS wide? Normally in adults — the QRS should *not* be more than *half* a large

box in duration (= not more than 0.10 second).

- **Regularity** — Look to see IF the rhythm is regular?
- Determine if P waves are "**Related**" to QRS complexes (ie, *conducting*) — Look *in front* of each QRS to see IF there is a P wave with a **fixed PR** interval.
- **Rate** — is most easily calculated by the **Rule of 300** (*Section 02.2*).

02.2 – Calculating Rate: The Rule of 300

Provided that the rhythm is regular — the **Rule of 300** states that heart rate can be *estimated IF* you **divide 300** by the **number of large boxes** in the **R-R interval**. The rule is derived as follows:

- With the ECG machine set at the standard recording speed of 25 mm/second — the time required to record each **little box** on ECG grid paper is **0.04 second** (*Figure 02.2-1*). Vertically — each *little box* is 1 mm in amplitude (*a measurement we will use often in assessing for chamber enlargement*).
- As seen in *Figure 02.2-1* — the time required to record each **large box** on ECG grid paper is **0.20 second**. That is — there are **5 little boxes** in each **large box**, and $5 \times 0.04 \text{ second} = 0.20 \text{ second}$ (= 1/5th of a second).

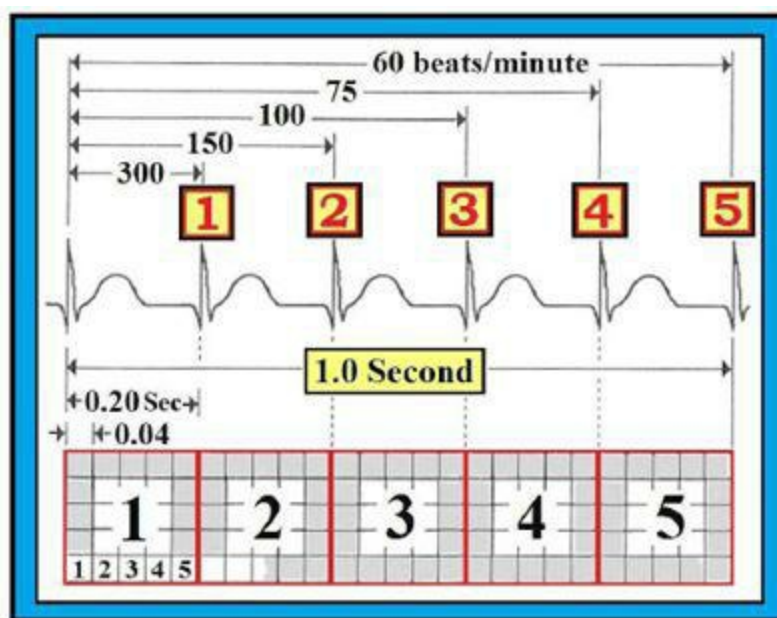


Figure 02.2-1: Deriving the "**Rule of 300**" (*See text*).

Thus, the time required to record **5 large boxes** will be one *full* second ($0.20 \times 5 = 1.0 \text{ sec}$). IF a QRS complex occurs each large box (*as in Figure 02.2-1*) — then the R-R interval will be 0.20 second — and the **rate** of the rhythm is **300 beats/minute** (ie, *5 beats occur each second X 60 seconds/minute* = a rate of 300/minute).

- IF the R-R interval is **2 large boxes** — then the **rate** will be *half* as fast (ie, $300 \div 2 = 150 \text{ beats/minute}$).

- IF the R-R is **3** boxes, the rate = **100/minute** ($300/3$).
- IF the R-R is **4** boxes, the rate = **75/min** ($300/4$) ...

NOTE: — Use the *every-other-beat method* when the rhythm is regular and the rate is fast (See Section 02.31).

02.3 – How to Define Sinus Rhythm?

By definition (*under normal circumstances, assuming the heart lies in the left side of the thorax*) — the **P wave** should always be *upright* in standard **lead II** when the mechanism of the rhythm is **sinus**. This is because the overall direction of the electrical depolarization wavefront as it travels from the SA node toward the AV node and the ventricles is oriented *toward* standard lead II, which lies at +60 degrees in the frontal plane (**Panel A** in **Figure 02.3-1**).

- The only 2 exceptions to the above stated rule (ie, *when the rhythm may still be sinus despite a negative P wave in lead II*) are: **i**) if there is dextrocardia; or **ii**) if there is lead misplacement.

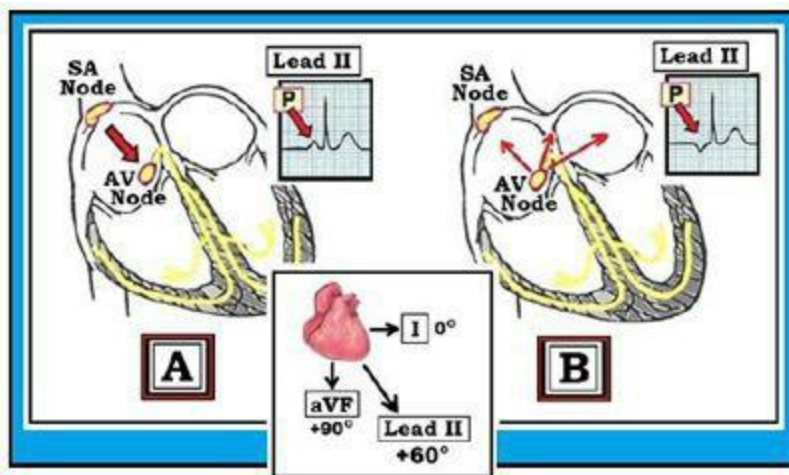


Figure 02.3-1: IF the rhythm is **sinus** — then the P wave should *always* be *upright* in lead II (**Panel A**). The only exception is IF there is *dextrocardia* or lead misplacement. Therefore — **Panel B** is *not* a sinus rhythm (See text).

In contrast to the situation with sinus rhythm — the P wave will *not* be positive (*upright*) in lead II when the electrical impulse originates from the AV node (**Panel B** in **Figure 02.3-1**). Instead — spread of atrial activation is now directed *away* from lead II.

- Therefore, with junctional beats or junctional (*AV nodal*) rhythm — a **negative P wave** may be seen to precede the QRS complex in **lead II** (**Panel B** in **Fig. 02.3-1**).
- Alternatively (*with AV nodal rhythm*) — a negative P wave may *follow* the QRS or *no P wave at all* is seen in lead II.

02.4 – FIGURE 02.4-1: Is the Rhythm Sinus?

Interpret the 3 rhythm strips shown in **Figure 02.4-1**.

- How can you tell at a glance IF the rhythm is sinus? (*Assume no dextrocardia and no lead misplacement*).

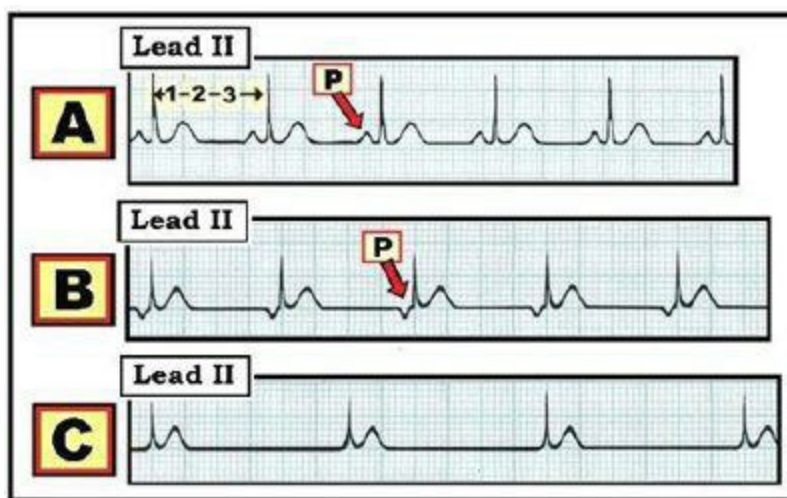


Figure 02.4-1: How to tell IF the rhythm is sinus?

Answer to Figure 02.4-1: The very *1st thing* we do when assessing any cardiac rhythm or 12-lead ECG is to look for a lead II rhythm strip. It takes *no more* than 2 brief seconds to **look in front of each QRS in lead II**.

- **Panel A** (in Figure 02.4-1) — Regular *upright* P waves precede each QRS with *fixed* PR interval in this lead II. Therefore — there is **NSR** (*N*ormal *S*inus *R*hythm). The QRS is narrow (*not more than half a large box*) — and the ventricular rhythm is regular with an R-R interval just *under* 4 large boxes in duration. IF the R-R interval was precisely 4 large boxes in duration — then by the **Rule of 300** the rate would be $300/4 = 75/\text{minute}$ (*Section 02.2*). Since the R-R interval is slightly *less* than 4 large boxes in duration — the rate must be slightly *faster* than 75/minute, or about **78/minute**.
- **Panel B** (in Figure 02.4-1) — does *not* represent sinus rhythm. It can't (*provided there is no dextrocardia or lead misplacement*) — since the QRS in lead II is *not* preceded by an upright P wave. Whether the rhythm in Panel B represents AV nodal or low atrial rhythm is *less* important. The **KEY** point — is that the rhythm is *not* sinus!
- Otherwise — the QRS is narrow and the rhythm is regular with an R-R interval of 4 large boxes in duration. Therefore — the rate = $300/4 = 75/\text{minute}$. The *negative* P waves in this lead II are conducting — because the PR interval preceding each QRS is fixed!
- **Panel C** (in Figure 02.4-1) — is *not* a sinus rhythm (*since no P wave at all is seen*). The QRS is narrow and the rhythm is regular with an R-R interval of 6 large boxes. Therefore, the rate = $300/6 = 50/\text{minute}$. This is an **AV nodal rhythm** (*See Sections 02.32-to 02.34*).

02.5 – Sinus Mechanism Rhythms/Arrhythmias

Once the mechanism of the rhythm is defined as “sinus” — the rate (*and regularity*) of the rhythm determine our terminology. There are 4 principal *sinus* mechanism rhythms (**Figure 02.5-1**):

- **NSR** (*Normal Sinus Rhythm*) — regular rhythm with a rate *between* 60-99/minute in an adult (**Panel A** in **Figure 02.5-1**). Norms for rate differ in children (*Section 02.6*).
- **Sinus Bradycardia** — regular sinus rhythm at a rate *below* 60/minute (**Panel B**). The rate here is 50/minute.
- **Sinus Tachycardia** — sinus rhythm at a rate of 100/minute or *faster* in an adult (**Panel C** in **Fig. 02.5-1**).
- **Sinus Arrhythmia** — an *irregular* rhythm *despite* the presence of a sinus mechanism (**Figure 02.7-1** — *Section 02.7*).

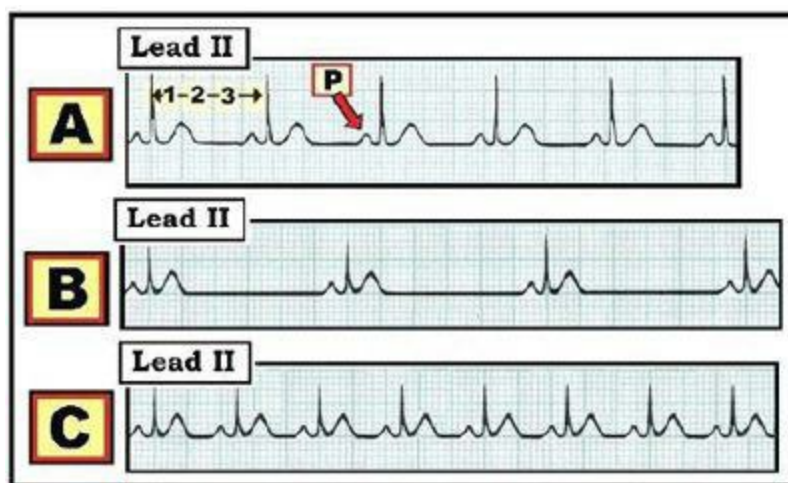


Figure 02.5-1: Sinus mechanism rhythms (*See text*).

02.6 – Norms for Children: *Different than Adults*

Normal limits for heart rate are *different* in children. While we primarily concern ourselves with the adult ECG in this book — it is nevertheless helpful to be aware of some differences between pediatric and adult findings.

- Rate limits are ***different in children*** — with “norms” depending on the age of the child. Much more important than specific “cut-off” points for each age category — is appreciation of the general concept that *slightly* faster rates (*say ~110/minute*) may be normal for a crying but otherwise healthy young child. By the same token — a rate within the “seemingly normal” range of 65/minute might be relatively *slow* for an active young child. *Clinical context is everything!*
- Upper limits for **PR** and **QRS interval duration** are also different (*shorter*) in the *smaller* pediatric heart (*Section 04.3*).
- Similarly — “***Escape***” rate ranges differ in children. Whereas the normal **AV nodal escape rate** in an **adult** is ***between 40-to-60/minute*** — it is *between 50-80/minute* in a young child. Therefore, the **AV nodal rhythm** at **75/minute** seen below in **Figure 02.6-1** would be slightly *fast* IF the patient was an adult — but is clearly *within* the normal AV nodal escape rate range IF the patient is a child (*See also Sections 02.32-to-02.34 on AV nodal rhythms*).



Figure 02.6-1: AV nodal rhythm at 75/minute (*See text*).

02.7 – Sinus Arrhythmia

Sinus Arrhythmia is a **common normal variant** in infants and young children. At times, variability in sinus rate may be marked — in which case one might initially think *some other* arrhythmia is present.

- **Sinus Arrhythmia** — is confirmed by the finding of *identical-appearing* P waves that are *upright* in lead II with *fixed* PR interval (**Figure 02.7-1**). In contrast — *other* phenomena (*wandering atrial pacemaker*; *sinus rhythm with PACs*) will manifest changes in P wave morphology *and/or* in the PR interval. *The P waves will be different!*



Figure 02.7-1: Sinus Arrhythmia. Despite *irregularity* in the rhythm — sinus mechanism is present as defined by regularly-occurring *upright* P waves with *fixed* PR interval preceding *each* QRS complex in this lead II rhythm strip (*See text*).

- Sinus arrhythmia often exhibits **respiratory variation**. This is especially true in healthy young children — for whom some variation in sinus regularity is the norm rather than the exception. Sinus “arrhythmia” is a *normal* cardiac rhythm in this setting.
- Some degree of sinus variability (ie, *sinus arrhythmia*) may persist in young *and even* older adults. This is *not* necessarily abnormal. *Clinical correlation is key*.

Beyond-the-Core: The **technical definition** of sinus “**arrhythmia**” — is that *sinus-initiated* R-R intervals **vary** by **at least 0.08-0.12 second** (2-3 little boxes). This definition is clearly satisfied in **Figure 02.7-1**. That said, most of the time — the presence of sinus “arrhythmia” (vs *sinus rhythm*) is of *little-to-no clinical* significance.

- An **exception** to the statement that sinus arrhythmia is benign occurs in *older* patients with **SSS** (Sick Sinus Sndrome). Among the many manifestations of SSS (*which include sinus pauses, sinus arrest, tachy- as well as bradyarrhythmias*) — **sinus bradycardia with sinus arrhythmia** is the most common. The usual course of SSS is prolonged over years (*if not decades*). Many patients who go on to develop full-fledged symptoms (*with need for a permanent pacemaker*) manifest *no more* than sinus bradycardia/sinus arrhythmia for a period of many years. Therefore — the finding of an *inappropriately* slow and *variable* sinus rhythm in an older patient with

symptoms of fatigue, worsening heart failure *and/or* syncope/presyncope is cause for potential concern. IF not due to rate-slowng drugs, ischemia or hypothyroidism — *Consider SSS* as the probable cause.

02.8 – FIGURE 02.8-1: *What Happens to the P in Lead II?*

Although specifics of the rhythm diagnosis in Figure 02.8-1 are advanced — the concept of routinely **seeking out a long lead II** rhythm strip *each* time you interpret a 12-lead ECG is fundamental! *Always begin with this step.*

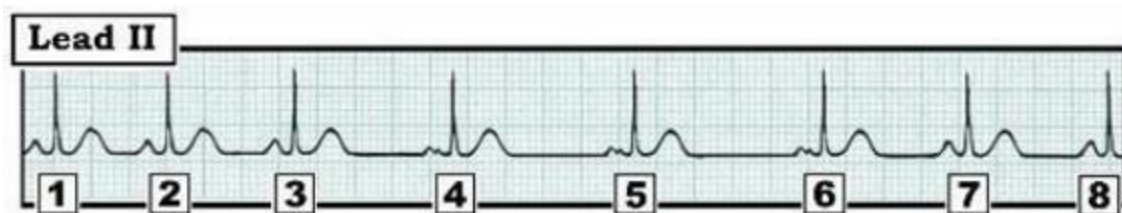


Figure 02.8-1: *What happens to the P wave in this lead II rhythm strip? (See text).*

Answer to Figure 02.8-1: Use the **Ps, Qs & 3R Approach** to assess the rhythm. As noted Section 02.1 — it does not matter in what *sequence* you look at the 5 parameters (*as long as you check them all each time you assess an ECG*):

- The overall rhythm in Figure 02.8-1 is *not* regular.
- The QRS complex is narrow.
- Each QRS is preceded by a P wave — BUT the *shape* of these P waves is not the same. The rhythm begins with a taller *peaked* P wave with fixed PR interval preceding beats #1,2,3; and #7,8. A 2nd P wave shape (*of smaller amplitude and notched*) — is then seen to precede beats #4,5,6. Thus, the **underlying rhythm** in Figure 02.8-1 is **sinus arrhythmia** — with *transient* change to *another* atrial focus for beats #4,5,6 — followed by *resumption* of the original sinus focus at the end of the tracing.

Key Points about Figure 02.8-1: In our experience — the most common error in 12-lead ECG interpretation is *failure* to recognize when normal sinus rhythm is not present. The solution is easy:

- Ideally — there will be a *simultaneously* recorded **long lead II** rhythm strip. IF so — *Begin* by:
i) assessing IF the rhythm is regular; and ii) Looking to see IF P wave morphology *stays the same* throughout the rhythm strip.
- It suffices to recognize that the *underlying* rhythm in Figure 02.8-1 is sinus with a *change* in P wave morphology. It is *easy* to see this as long as you *consciously* look at P wave shape before *each* QRS.
- It is clearly more difficult *without* a long lead rhythm strip. How would you *assess the rhythm* in Figure 02.9-1?

02.9 – FIGURE 02.9-1: *When there is NO long Lead II Rhythm Strip ...*

The reality is — that you will *not* always have access to a long lead rhythm strip. This was the case for the 12-lead ECG previously shown in [Figure 01.3-1](#) (*Section 01.3*). We reproduce that 12-lead tracing below in [Figure 02.9-1](#):

- How would you assess the rhythm in Figure 02.9-1?
- **How many *total* beats** are there on this 12-lead ECG?

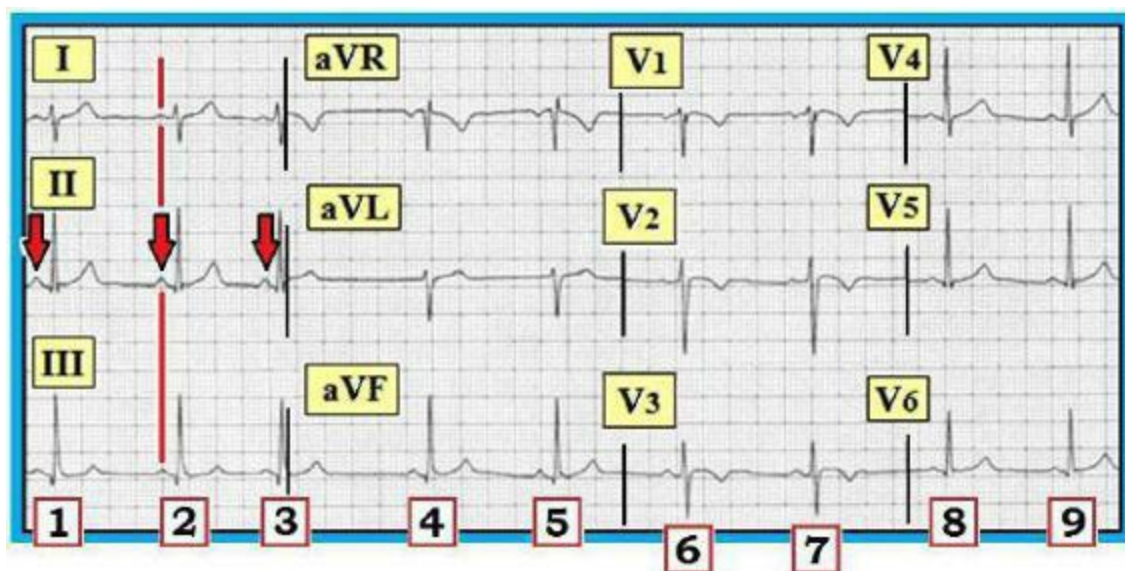


Figure 02.9-1: 12-lead ECG previously shown in [Figure 01.3-1](#). How many *total beats* are there on this tracing? What is the rhythm? Unfortunately — there is no long lead II rhythm strip (*See text*).

Answer to Figure 02.9-1: The problem is that *only* 3 beats are seen in lead II of this ECG (*arrows*) before the leads switch to *simultaneous* recording of leads aVR/aVL/aVF. In addition — the R-R interval for these 3 beats that are seen in lead II is *not* the same (ie, *the R-R interval between beats #1-2 is longer than the R-R between beats #2-3*). That said, we can still diagnose the ***mechanism*** as **sinus** — because a *similarly-shaped* upright P wave with *fixed* PR interval precedes *each* of these 3 beats in lead II (*red arrows in Figure 02.9-1*).

- There are **9 total beats** on this tracing. With *simultaneous* 3-channel recordings — we get “3 looks” at each beat (*vertical time line in leads I – II – III*). The QRS is narrow. Note *irregularity* of the R-R interval for beats #1-*thru*-9. Thus — the rhythm is **sinus arrhythmia**.

02.10 – **Advanced POINT:** *What is a Wandering Pacemaker?*

Occasionally — the site of the atrial pacemaker may shift (*wander*) away from its usual site of origin in the SA (*Sino-Atrial*) Node. In most cases — **wandering atrial pacemaker** is a benign normal variant that occurs in patients *without* underlying heart disease. It may result from *variations* in vagal tone (*that slow SA nodal discharge and allow other atrial sites to temporarily emerge*) — or there may be no obvious cause ([Figure 02.10-1](#)).

- **ECG recognition** of wandering pacer requires a *long enough* rhythm strip to appreciate **gradual change** over a period of beats from one P wave morphology to another. While possible that the rhythm in Figure 02.8-1 could reflect a wandering pacemaker — this rhythm strip is simply *not long enough* to tell.

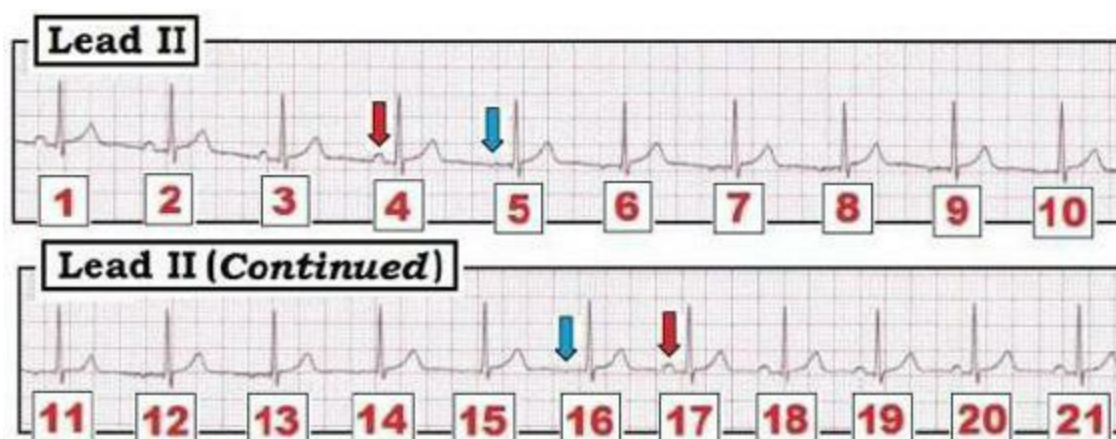


Figure 02.10-1: Wandering pacemaker. Initially the P wave in lead II is an upright sinus complex (*1st red arrow preceding beat #4*) — with **gradual change** to an isoelectric P wave (*1st blue arrow preceding beat #5*) — followed by resumption of sinus rhythm with beat #17 (*2nd red arrow in bottom rhythm strip*). It would be *easy-to-overlook* the diagnosis of wandering pacer IF one did not pay *careful* attention to P wave morphology!

Bottom Line: True *wandering atrial pacemaker* is *not* a common diagnosis. As noted — it is difficult to recognize because most 12-lead ECGs (*even if accompanied by a simultaneous lead II rhythm strip*) will simply *not* be long enough to manifest gradual transition *back-and-forth* between SA node and two or more additional atrial sites. The point to emphasize for interpreters of *any* experience level — is the need to *always* **look carefully** at **lead II** when assessing for *atrial* activity:

- Is an *upright* sinus P wave present in lead II?
- Does P wave morphology *change* during the rhythm?
- Is the underlying rhythm regular? Are there any *early* beats?

02.11 – FIGURE 02.11-1: *Why is this NOT Wandering Pacer?*

While *not* yet worrying about the specific diagnosis — Why is wandering pacer *not* present in Figure 02.11-1?

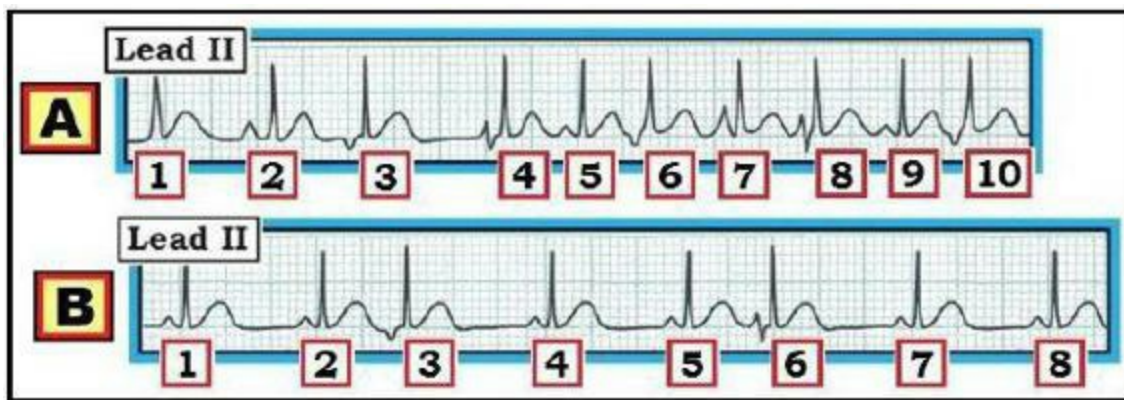


Figure 02.11-1: Why is wandering pacemaker not present in either of the rhythm strips shown here?

Answer to Figure 02.11-1: The rhythm in **Panel A** is MAT (See Section 02.16). There are too many different P waves that change from *beat-to-beat* for this to be wandering pacer. In **Panel B** — the underlying rhythm is sinus. Beats #3 and #6 are PACs; they occur *early* — and P shape is different (See Section 02.56).

- True *wandering* pacemaker should manifest *gradual* transition from one P wave morphology to another — and then *either* on to yet another atrial pacemaker site or *back* to the original focus. This *gradual* transition was seen in Figure 02.10-1. It is definitely not seen in either Panel A or B of Figure 02.11-1.

02.12 – Other Supraventricular Rhythms

A *supraventricular* rhythm is defined to be one in which the electrical impulse originates at or above the AV node (at or above the double dotted line in **Figure 02.12-1**).

- The “good news” — is that the **number of sites** from where a *supraventricular* impulse may arise is **limited**. These include: **i)** the SA Node; **ii)** from *somewhere* in the atria; or **iii)** the AV node. Awareness of this clinical reality greatly facilitates arrhythmia diagnosis *because* once you establish that the QRS complex in an adult is **truly narrow** in all 12 leads — you are virtually *assured* that the rhythm is supraventricular (>99% of the time)!
- The exception (occurs 0.1% of the time) — in which a *narrow* QRS may arise from a *ventricular* site in an adult is IF the impulse originates from the His, fascicles or bundle branches.
- The above generality does *not* hold true in children (*the QRS is normally narrower in the smaller pediatric heart*).

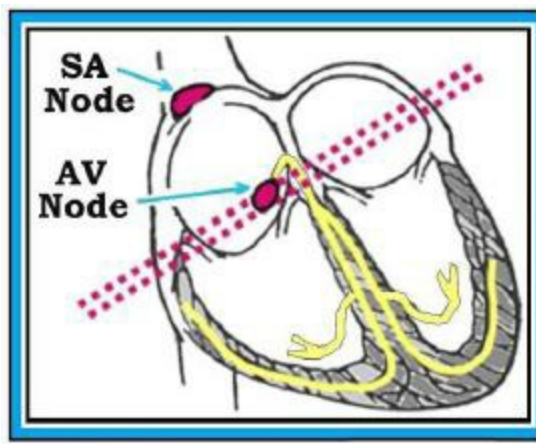


Figure 02.12-1: IF the QRS is *truly* narrow in *all* 12 leads — then for practical purposes the rhythm is **supraventricular** (and therefore originates from at or above the double dotted red line in this Figure). **NOTE:** The opposite is *not* true. That is — the etiology of a rhythm is *not* at all certain **IF** the QRS is wide (could be either ventricular or supraventricular — See Figure 02.13-1).

Other Supraventricular Rhythms: In addition to the **sinus mechanism rhythms** (Section 02.5) — the **other** principal **entities** in the **supraventricular rhythm category** include:

- AFib (*Atrial Fibrillation*) — See Section 02.14.
- MAT (*Multifocal Atrial Tachycardia*) — See Section 02.16.
- AFlutter (*Atrial Flutter*) — See Section 02.18.
- PSVT/AVNRT — See Section 02.29.
- Junctional (*AV nodal*) rhythms — See Sections 02.32-to-02.34.

NOTE: Although there are *many* types of **supraventricular rhythms** — those listed above make up the great *majority* of rhythms that primary care and emergency providers most often will see.

- Unless there is *preexisting* bundle branch block or *aberrant* conduction — the **QRS** will be **narrow** **IF** the rhythm is supraventricular.

02.13 – FIGURE 02.13-1: *Why is this Rhythm Supraventricular?*

Despite the slow rate and very wide QRS complex — *WHY* is the rhythm in Figure 02.13-1 supraventricular?

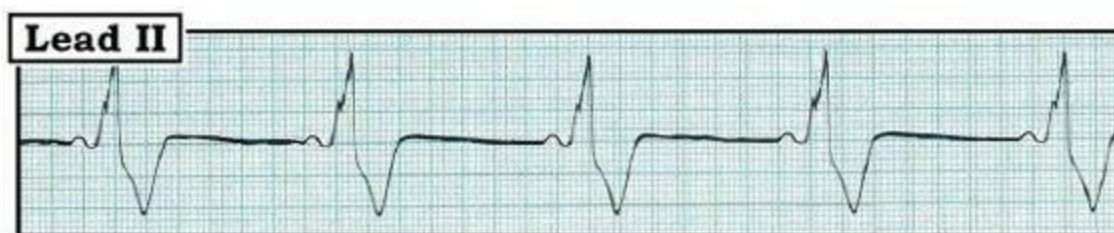


Figure 02.13-1: Despite the wide QRS — Why is this rhythm supraventricular?

Answer to Figure 02.13-1: By the Ps, Qs & 3R Approach — We interpret the rhythm in Figure 02.13-1 as follows:

- Regular *wide* QRS rhythm at ~40/minute.
- *Upright* P waves precede each QRS complex with a *fixed* and normal PR interval in this lead II rhythm strip. Therefore — the rhythm is **sinus bradycardia** with **QRS widening** as a result of **preexisting BBB** (*B*undle *B*ranch *B*lock).

02.14 – Atrial Fibrillation

AFib (*A*trial *F*ibrillation) — is characterized by the presence of an **irregularly irregular** rhythm in the *absence* of P waves (**Figure 02.14-1**). Undulations in the baseline (known as “*fib*” waves) may sometimes be seen. Such “fib waves” are *coarse* in **Tracing B** of Fig. 02.14-1 — *fine* in **Tracing C** — and are *barely detectable* in **Tracing A**.

- As might be imagined — the diagnosis of AFib is clearly more difficult when “fib waves” are of minimal amplitude (in which case diagnosis is based solely on recognizing an irregularly irregular rhythm in the absence of P waves).



Figure 02.14-1: Atrial fibrillation (See text).

KEY Clinical Point: Far better than simply saying the rhythm is “AFib” — is to **clarify** the **ventricular response**. Thus, AFib will conduct with either a rapid, controlled, or slow ventricular response:

- **AFib with a rapid ventricular response** is present — IF the *average* rate is *over* ~120/minute (**Tracing A** in Fig. 02.14-1). *New-onset* AFib is most often rapid (*unless* slowed by drugs, ischemia/infarction or sick sinus syndrome).
- **AFib with a controlled ventricular response** is present — IF the *average* rate of AFib is *between* ~70-110/minute (**Tracing B** in Fig. 02.14-1). An important goal of medical treatment is to *attain* rate control.
- **AFib with a slow ventricular response** is present — IF the *average* rate is *less* than 50-60/minute (**Tracing C**). *New-onset slow* AFib is unusual — and should suggest a different set of

diagnostic consideration (*rate-slowing drugs; hypothyroidism; ischemia/infarction; sick sinus*).

Clinical PEARL: When AFib is rapid — the irregularity in the rhythm may be subtle. As a result — *rapid* AFib may sometimes *simulate* PSVT. We see this toward the end of **Tracing A** (in **Fig. 02.14-1**).

- **Calipers** may be needed to verify that the rhythm is in fact irregular *throughout* the tracing.

02.15 – Advanced POINT: Very Fast AFib — Think WPW!

The usual rate range for “*rapid*” A Fib is *between* ~110-180/minute. IE ever the rate of A Fib is significantly *above* this (ie, >200-220/minute) — the patient probably has an AP (Accessory Pathway) that is *bypassing* the AV node (ie, **WPW** = Wolff-Parkinson-White Syndrome).

- The intrinsic *refractory period* of the normal AV node generally does *not* allow conduction of more than 200-220 impulses per minute.

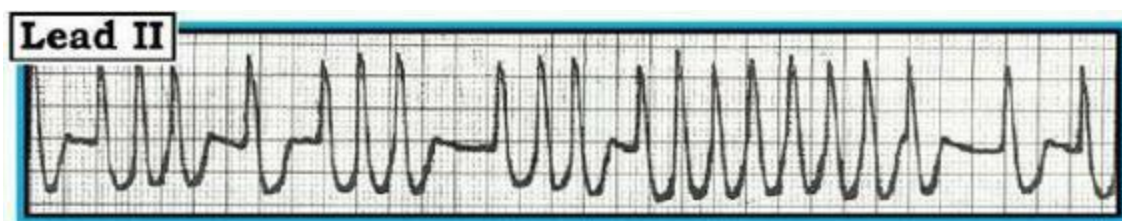


Figure 02.15-1: The QRS complex is wide. **AFib** is diagnosed by the *irregular* irregularity in the *absence* of P waves. The rate attains 250-300/minute in parts of the tracing. This is virtually diagnostic of **WPW** (See Section 05.49).

02.16 – Multifocal Atrial Tachycardia

AFib should be distinguished from **MAT** (Multifocal Atrial Tachycardia) — in which the rhythm is also *irregularly* irregular, but in which *definite* P waves are present (*arrows* in **Figure 02.16-1**). Note that P wave morphology changes from *beat-to-beat* with MAT — which is what distinguishes MAT from wandering atrial pacemaker (See **Figure 02.10-1**). The name *tells all* about its ECG appearance: Multiple Atrial foci (P waves) are seen at a *fast* rate (ie, **Tachycardia**).

- In our experience — **MAT** is the 2nd most commonly overlooked arrhythmia diagnosis (*next to AFlutter*). MAT is **easy to overlook** — because the overwhelming majority of sustained *irregular* SVT rhythms are AFib.
- Clinically — MAT is most often seen in patients who have either: **i)** *pulmonary* disease or **ii)** severe *multisystem* problems (*sepsis, shock, acidosis, electrolyte abnormalities, etc.*). The *best* way to avoid overlooking MAT is to **think of this diagnosis whenever** you see an *irregularly irregular* rhythm in either of the above settings.
- **12 leads** are *better* than one. You may *not* always see different P wave shapes in a single

monitoring lead. Get a **12-lead ECG** and look for atrial activity in *all* 12 leads.



Figure 02.16-1: MAT is distinguished from AFib by the presence of multiple *different-looking* P waves. The importance of recognizing MAT is its different treatment (*Section 02.17*).

02.17 – FIGURE 02.17-1: *Why is this Not AFib?*

Why is the *irregularly* rhythm seen in Figure 02.17-1 not AFib? HINT: This ECG was obtained from a patient with COPD (*Chronic Obstructive Pulmonary Disease*).

- Can you see atrial activity in *some* of the 12 leads? If so — in *which* ones?
- Clinically — Why is it important to make the correct rhythm diagnosis?

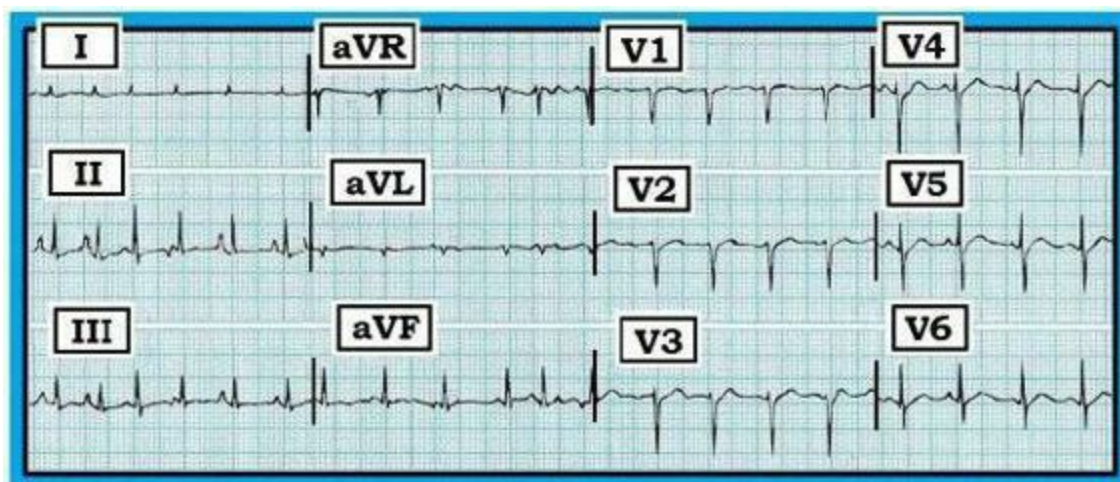


Figure 02.17-1: What is the rhythm on this 12-lead ECG? *What else* do you see on this tracing that supports your rhythm diagnosis? 152

Answer to Figure 02.17-1: The rhythm is *irregularly* irregular. The QRS complex is narrow. The rhythm in Fig. 02.17-1 is not AFib — because P waves are clearly seen in lead II. P waves are also seen in leads III and aVF — but not in other leads.

- **P wave morphology** in lead II *changes* from beat to beat. This observation plus knowing this patient has **COPD** helps to *solidify* the ECG rhythm diagnosis of **MAT**.
- *Additional* support of the diagnosis of MAT is forthcoming from recognition of several **ECG findings** that suggest **significant pulmonary disease**. These include: i) Schamroth's sign (*very low QRST amplitude in lead I*) ; ii) tall *pointed* P waves in lead II among the P wave morphologies seen; and iii) persistence of precordial S waves through to V5,V6 (*See Section 08.33*).

- The clinical importance of recognizing MAT (*and distinguishing it from AFib*) — is that treatment of these two entities is different! **Treatment of MAT** — is *best* directed at correcting the *underlying cause* (*optimizing oxygenation in this case*). In contrast — Treatment of **AFib** focuses on rate control; converting the rhythm; and anticoagulation considerations.

KEY Clinical Point: Rather than a discrete entity — think of MAT as *part* of a **spectrum**. At one end — is sinus rhythm with *occasional* PACs (**Tracing B** in Figure 02.11-1). At the other — is MAT in which P wave morphology (*and the PR interval*) change from *beat-to-beat*. Along the way you may see sinus rhythm with *multiple* PACs (*but not quite enough beat-to-beat change to diagnose “MAT”*).

- Clinically — implications of the various arrhythmias that may be seen *throughout* this spectrum are similar. These are to diagnose and treat the underlying disorder as first priority (*usually hypoxemia from pulmonary disease; electrolyte or acid-base disturbance; other metabolic problems*).

02.18 – Atrial Flutter

AFlutter (*Atrial Flutter*) — is characterized by a special pattern of *regular* atrial activity that in adults *almost always* (*and almost magically*) occurs at a rate of **300/minute** (*250-350/minute = usual range*). Atrial flutter typically manifests a **sawtooth** appearance that is usually best seen in the *inferior* leads — although at times, flutter waves may be subtle and only seen in a few leads (**Figure 02.18-1**).

- The **most common** *ventricular* response to atrial flutter (*by far!*) is with **2:1 AV conduction**. As a result, the ventricular rate with **untreated** atrial flutter will usually be *close* to **150/minute** (ie, $\sim 300 \div 2$). **NOTE:** The atrial rate *may* slower IF antiarrhythmic drugs are used.
- Less commonly with AFlutter there is **4:1 AV conduction** (*ventricular rate $\sim 75/\text{minute}$*) — or a **variable** (= *irregular*) **ventricular response**.
- **Odd ratios** (ie, *1:1, 3:1, 5:1*) are possible but **extremely uncommon** (*unless the patient has WPW or is already on antiarrhythmic drugs*).

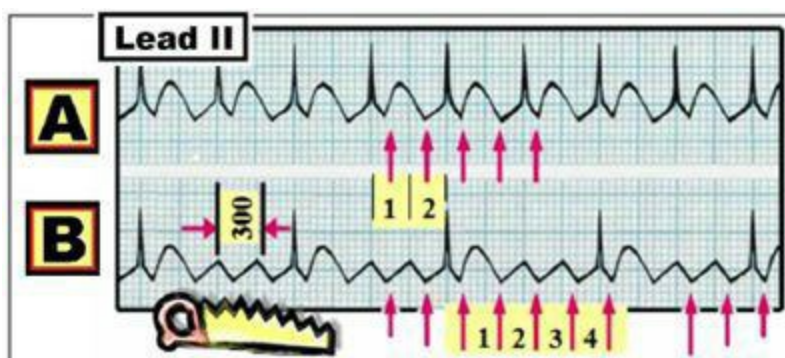


Figure 02.18-1: Atrial Flutter with 2:1 AV conduction (**Tracing A**). Note how much easier it is to identify AFlutter when the AV conduction ratio is slower (*as it is with 4:1 AV conduction in **Tracing***

B). Tracing B shows the effect that a **vagal maneuver** might have (See Sections 02.26, 02.27).

02.19 – FIGURE 02.19-1: *Easy to Overlook AFlutter ...*

In our experience — **AFlutter** is by far the **most commonly overlooked rhythm diagnosis**. It is *easy* to overlook — because flutter waves may not always be evident in the lead you are monitoring.

- **Always suspect AFlutter** (until proven otherwise) — whenever there is a **regular SVT** at **~150/minute** without clear sign of normal atrial activity. This is precisely what is seen in (**Figure 02.19-1**). The patient was stable.

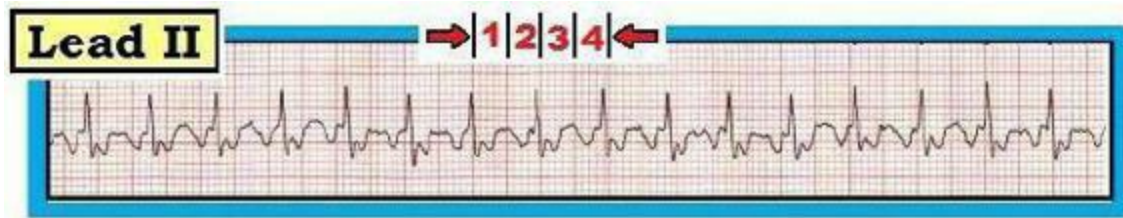


Figure 02.19-1: There is a **regular SVT** (*narrow QRS rhythm*) at a rate of **~150/minute**. Normal atrial activity is not seen (*no clear upright P wave is evident in this lead II monitoring lead*). IF the patient is stable — Look for atrial activity in *other* leads!

IF the patient is *hemodynamically* stable and tolerating the tachycardia — then a **look** at **additional leads** may facilitate diagnosis:

- A **12-lead ECG** is obtained (**Figure 02.19-2**). Note how this helps to verify our suspicion of *non-sinus* atrial activity (*red and blue arrows*).

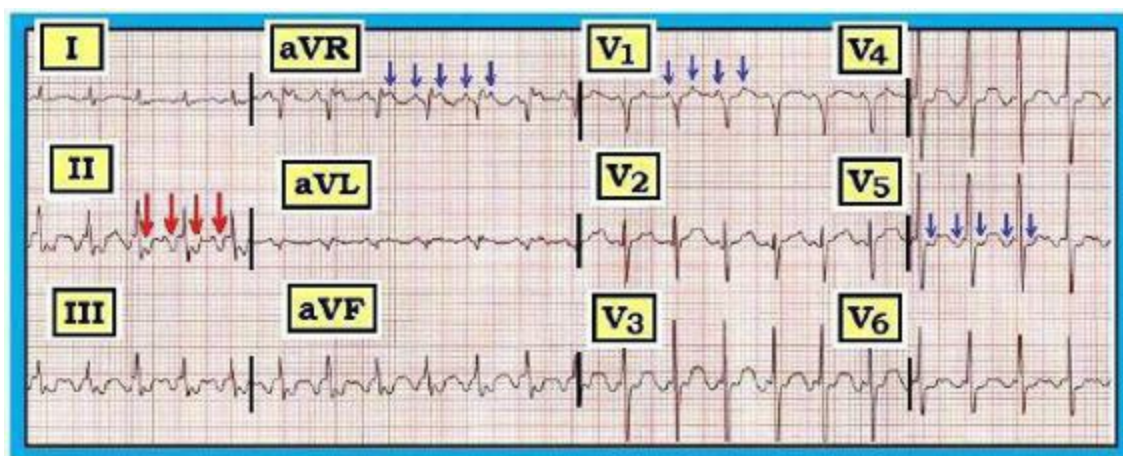


Figure 02.19-2: 12-lead ECG obtained on the patient whose initial rhythm was shown in **Figure 02.19-1**.

02.20 – *How NOT to Overlook AFlutter* (Figure 02.19-1)

We were not initially certain of the rhythm diagnosis in **Figure 02.19-1**. We assessed this rhythm as a **regular narrow-complex tachycardia** (ie, a *regular SVT*) at a rate of **~150/minute** without sinus P waves.

- We saw one (*if not two*) negative deflections *within* each R-R interval in Figure 02.19-1 suggesting atrial activity.
- Remembering the statement: “**AFlutter until proven otherwise**” for any regular SVT at ~150/minute *without* sinus P waves — We *suspected* this diagnosis (*but were not certain*).
- Since the patient was *hemodynamically* stable (*as they usually will be with SVT rhythms*) — We obtained a **12-lead ECG** (Figure 02.19-2). The 12-lead *confirmed* that the QRS was *truly* narrow in *all* leads. It also suggested *regularly* occurring atrial activity at *rapid* rate in *many* leads (*red and blue arrows in Figure 02.19-2*).

NOTE: There is *no* indication of atrial activity *during* the *regular* SVT in several leads in Fig. 02.19-2 (*leads I, aVL, V6*). Flutter waves are *not* always seen in all leads. That said — leads with the **arrows** clearly demonstrate that the deflections we see are “real”.

- **PEARL — Using Calipers:** Use of *calipers* greatly facilitates the diagnostic process. There is no easier way to identify if baseline deflections are regularly occurring or not. Using calipers enables us to diagnose AFlutter simply from the 12-lead tracing shown in Figure 02.19-2 (*arrows*).

02.21 – FIGURE 02.21-1: Vagal Maneuvers to Confirm AFlutter

Confirmation of AFlutter as the rhythm diagnosis for Figure 02.19-1 was forthcoming following application of a **vagal maneuver** (Figure 02.21-1).

- Note *transient* slowing of the ventricular response after the *large* arrow (*vagal maneuver*) — revealing **regular atrial activity** at ~300/minute. The only rhythm that does this is atrial flutter.
- Beyond-the-Core: Whether or not a vagal maneuver was truly needed in this case to confirm AFlutter vs *sufficient* comfort in this diagnosis from simply reviewing the 12-lead ECG in Figure 02.19-2 — would depend on the experience level, expertise and practice preferences of the treating clinician. Clinically — We would be *less* inclined to use IV adenosine once we *know* the rhythm is AFlutter (*since adenosine won't convert AFlutter*) — and *more* inclined to treat with an AV nodal blocking agent (*diltiazem, beta-blocker*) while contemplating more definitive measures.



Figure 02.21-1: A **vagal maneuver** is applied (*large arrow*) to the rhythm initially shown in Figure 02.19-1. The vagal maneuver temporarily *slows* AV nodal conduction — thereby revealing *underlying* atrial activity at 300/minute (*smaller red arrows*). The *only* rhythm that does this is

AFlutter.

02.22 – FIGURE 02.22-1: *Some KEY Aspects about AFlutter*

Given the elusiveness of AFlutter — We highlight some *advanced* special characteristics of this rhythm by the 4 tracings shown in **Figure 02.22-1**:

- How would you interpret each tracing in this Figure?
- Which concept regarding recognition/differential diagnosis of AFlutter do you think is illustrated by each example?

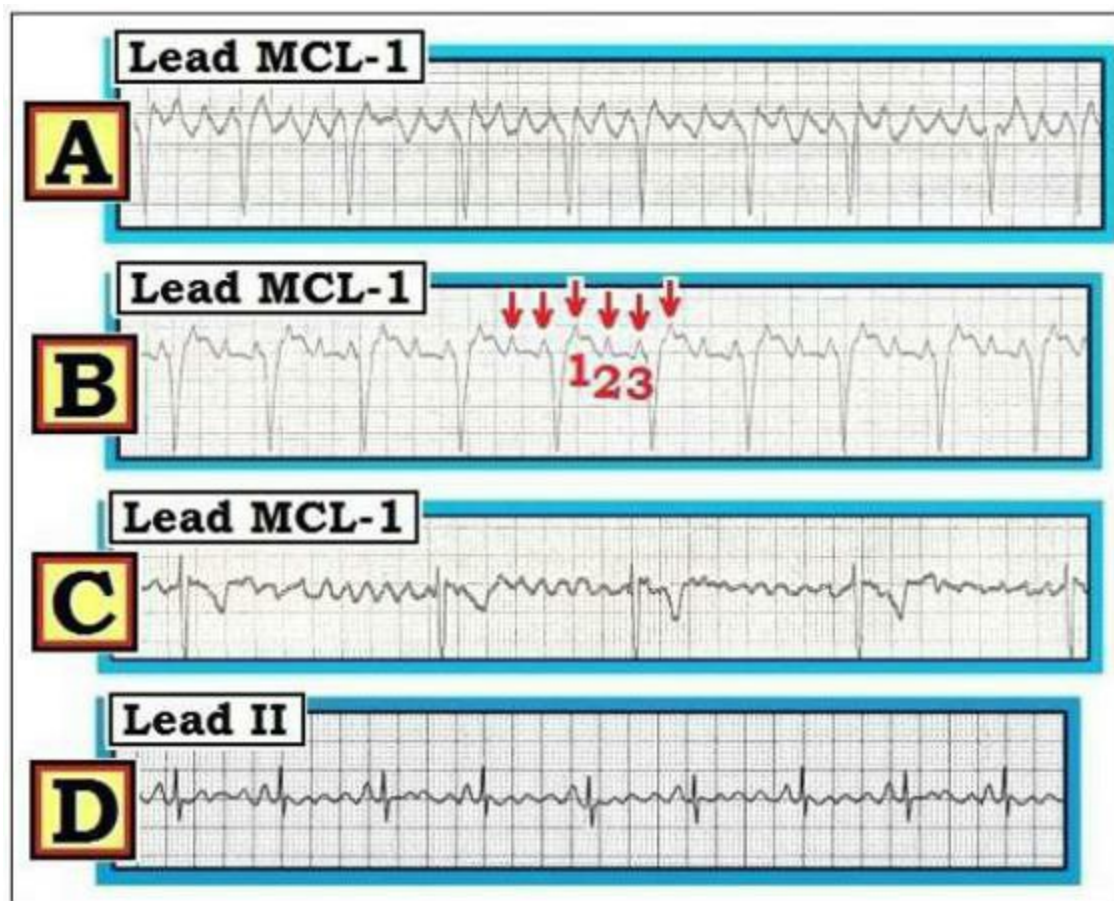


Figure 02.22-1 (Tracings A,B,C,D): How does each tracing relate to diagnosis or misdiagnosis of AFlutter?

ANSWER to Tracing A (in Figure 02.22-1):

- As noted in Section 02.18 — there may be a *variable* ventricular response to AFlutter (**Tracing A**). Despite *irregular* irregularity that would otherwise suggest AFib — the diagnosis of **AFlutter** is secure in Tracing A from the **precisely regular sawtooth pattern** of atrial activity seen here at a rate just over 300/minute.

02.23 – TRACING B: *AFlutter with 3:1 AV Conduction*



Figure 02.23-1: What is *unusual* about the example of AFlutter shown in Tracing B?

Answer to Tracing B:

- The rhythm in **Tracing B** (initially seen as B in Fig. 02.22-1) — is another example of **AFlutter**, this time with **3:1 AV conduction**. As noted in Section 02.18 — *odd* conduction ratios are *extremely* uncommon in AFlutter. That said — they *can* be seen on occasion. The diagnosis of **AFlutter** is secure in **Tracing B** from recognition of *precisely* regular atrial activity (*arrows*) at a rate of ~300/minute. *Nothing else does this*. We count 3 flutter waves within each R-R interval.

02.24 – TRACING C: AFib-Flutter

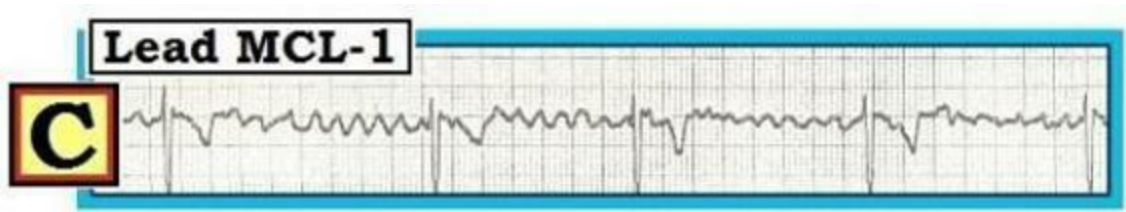


Figure 02.24-1: Is the rhythm in Tracing C AFib or AFlutter?

Answer to Tracing C:

- The rhythm in **Tracing C** (initially seen as C in Fig. 02.22-1) — shows a slow, *irregularly* irregular rhythm with narrow QRS and underlying atrial activity. Initially the rhythm looks like AFlutter — but toward the end of the tracing baseline undulations look more like AFib. Although often referred to as “**AFib-Flutter**” — the important clinical point is that the rhythm behaves as AFib. Although technically this rhythm is *best* classified as *very slow* AFib — We would *also* accept interpretation as *AFib-Flutter* as a *descriptive* term that perhaps most accurately conveys the ECG picture in Tracing C.

02.25 – TRACING D: AFlutter vs Artifact

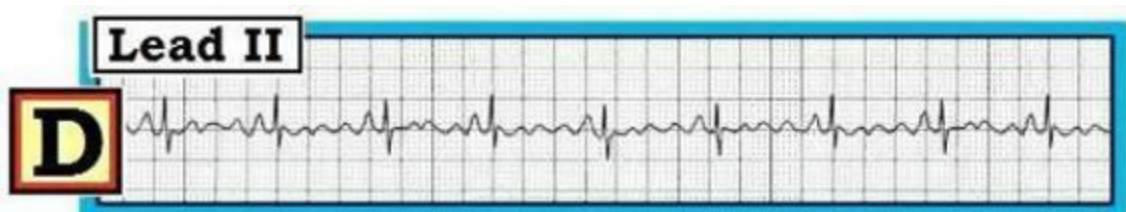


Figure 02.25-1: Is atrial activity in Tracing D real? What do you *suspect* the rhythm is? How might you confirm this?

Answer to Tracing D:

- The rhythm in **Tracing D** (*initially seen as D in Fig. 02.22-1*) — is *not* AFlutter. Instead — the numerous small undulations that populate the baseline represent **artifact**. We *know* the rhythm in **Figure 02.25-1** is not AFlutter because: **i)** We can see regular underlying P waves (*arrows in Figure 02.25-2*) ; **ii)** These P waves are *unaffected* by the baseline artifact; **iii)** the artifact undulations are too fast and irregular to be AFlutter; **and iv)** We looked at the patient (*who manifested an obvious resting tremor*).



Figure 02.25-2: Arrows highlight sinus P waves in Tracing D. These sinus P waves are superimposed on baseline artifactual undulations.

02.26 – Use of VAGAL Maneuvers (*Carotid Massage, Valsalva*)

Vagal maneuvers are commonly used to facilitate ECG diagnosis — *and/or* to treat certain cardiac arrhythmias. Vagal maneuvers work by producing a *transient* increase in **parasympathetic tone** — thus temporarily *slowing* conduction through the AV node. This was seen in Figure **Figure 02.21-1** — in which the vagal maneuver facilitated ECG diagnosis of AFlutter.

- **Carotid Sinus Massage (CSM)** — Always perform under *constant* ECG monitoring. Use the *right* carotid first. *Never* press on both carotids at the same time. Remember that the carotid sinus is located *high* in the neck at the angle of the jaw! (*green arrow in Figure 02.26-1*).



Figure 02.26-1: Carotid massage (*See text*).

Additional points to consider about CSM include:

- Warn patient that the maneuver will be uncomfortable (*as very firm pressure is needed for success*). Apply enough pressure to *indent* a tennis ball.
- Rub for no more than 3-5 seconds at a time.
- IF no response — may repeat CSM on the *left* side.
- *Don't* do CSM if patient has a carotid bruit (*as you may dislodge a carotid plaque*).
- **Always monitor** the patient on **ECG!** Mark the time you *start* carotid massage — and the time you stop (*which provides hard copy of what occurred*). ECG changes with CSM may be quite subtle (*which is another reason to get a hard copy rhythm strip recording during the time CSM is done*).

Valsalva — Have patient *supine*. Forcibly exhale (*bear down*) against a closed glottis (*as if trying to go to the bathroom*) for up to 15 seconds at a time. If properly performed — may be even *more* effective than CSM!

02.27 – FIGURE 02.27-1: Clinical Response to Vagal Maneuvers

Know the response you are looking for before beginning (**Figure 02.27-1**).

- At times — *reapplication* of a vagal maneuver *after* administration of an AV nodal blocking drug (ie, *diltiazem*; a β -blocker) may work, whereas it didn't work *before* the drug was given.
- A *similar* response as is described in Figure 02.27-1 would be expected with “**chemical**” **valsalva** (*from diagnostic/therapeutic use of Adenosine*).

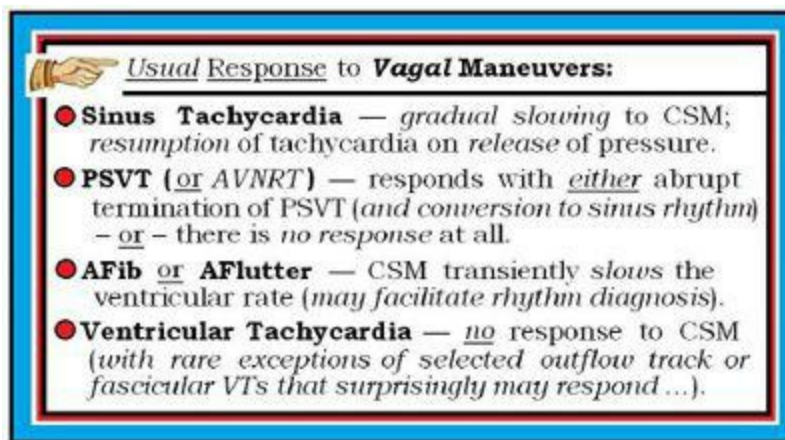


Figure 02.27-1: What to expect from CSM/Valsalva.

02.28 – Using ADENOSINE = “Chemical” Valsava

Adenosine is an extremely useful drug for treatment of SVT. It will *rapidly* convert *most* PSVT — and it usually produces transient *slowing* of other SVT rhythms which may be diagnostic (*as it is in Figure 02.28-1*).

- **Adenosine** may also convert 5-10% of VT (*Ventricular Tachycardia*) rhythms — especially outflow track VT that is prone to develop in younger adults without underlying heart disease. For this reason — *current* ACLS Guidelines recommend trial of Adenosine *early* in the course of treating either narrow or wide tachycardias of *uncertain* etiology.

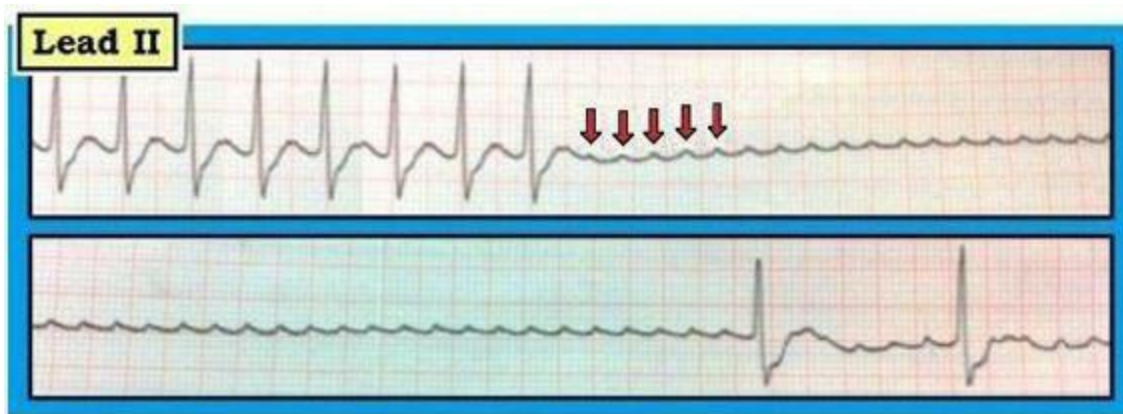


Figure 02.28-1: Administration of **Adenosine** (“chemical” *valsalva*) — was diagnostic of SVT etiology in this patient (*arrows reveal underlying AFlutter at ~300/minute*). But in so doing — a period of over 10 seconds ensued *without* a QRS complex. *Marked* bradycardia like this is *not uncommon* following Adenosine. Bradycardia almost always resolves *within* 30-60 seconds (*due to the drug’s ultra-short half-life*). Other *transient* side effects of Adenosine may include chest pressure; flushing; bronchospasm; metallic taste; a feeling of “impending doom”. (*Be sure to tell the patient these effects are short-lived!*).

NOTE: Fortunately — Using Adenosine *early* in the treatment of a *regular* WCT (*Wide-Complex Tachycardia*) of *uncertain* etiology is *unlikely* to be harmful (*given the ultra-short half-life of the drug*). The “good news” — is that doing so may convert *many* SVT rhythms — it will be *diagnostic* in others — and it may even convert a small percentage of VT rhythms.

- *Beyond-the-Core:* Adenosine is *unlikely* to work for VT with *wide* QRS in a patient with *ischemic* heart disease. As a result — we generally favor omitting it from the treatment protocol of such patients.
- Be aware that conversion of a *regular* WCT to sinus rhythm following use of Adenosine does *not* prove a *supraventricular* etiology (*since the drug may convert a small percentage of VT rhythms*).

02.29 – PSVT/AVNRT

PSVT (*Paroxysmal SupraVentricular Tachycardia*) — is a regular *supraventricular* tachycardia that most often occurs at a rate *between* 150-to-240/minute (**Figure 02.29-1**). Atrial activity is usually *not* evident — although subtle notching or a negative deflection (*representing retrograde atrial activity*) may sometimes be seen at the tail end of the QRS.

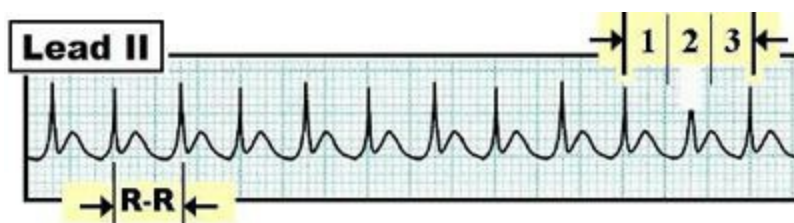


Figure 02.29-1: PSVT (*regular SVT without sinus P waves at ~200/minute*). Rate is calculated by the *every-other-beat* method (See Section 02.31).

Mechanistically — **PSVT** is a **reentry tachycardia** that involves at least some portion of the AV node (Figure 02.29-2). This accounts for the *other* name for this rhythm = **AVNRT** (**AV Nodal Reentry Tachycardia**).

- PSVT is often initiated by a PAC (*which arrives early at the AV Node — at a time when conditions are “just right” to allow the reentry circuit to be set up*). Once set up — the impulse *continues* to circulate *within* the AV Node (Figure 02-29-2) — *until* the reentry pathway is either interrupted (ie, by *AV nodal blocking drugs* or a *vagal maneuver*) — or until it stops *spontaneously*.
- Of note, each time the impulse circulates around the AV Node — it conducts a QRS *down* to the ventricles and also conducts *retrograde* back up to the atria (Fig. 02.29-2).

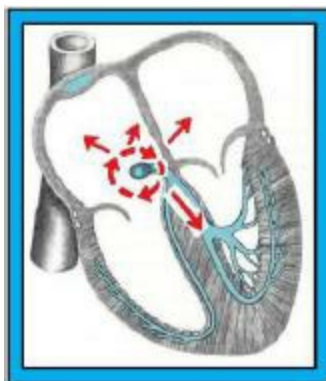


Figure 02.29-2: PSVT. *Schematic illustration of reentry involving a portion of the AV Node. Each time the impulse circulates around the AV node — it conducts a QRS *down* to the ventricles and also conducts *retrograde* back up to the atria.*

02.30 – FIGURE 02.30-1: **Retrograde Conduction with PSVT**

Most of the time, atrial activity will not be seen *during* PSVT — because the retrograde P wave will be *hidden* within the *simultaneously* occurring QRS complex.

- **Beyond-the-Core:** On occasion — you will see notching in the *terminal* portion of the QRS complex *during* PSVT (**Figure 02.30-1**). When present — this *retrograde* atrial activity indicates a **reentry mechanism** is operative as the mechanism of the SVT. Clinically — *reentry* SVT rhythms are more likely to respond to vagal maneuvers *and/or* use of Adenosine.
- **Beyond-the-Core:** Confirmation that notching in the *terminal* portion of the QRS complex truly represents *retrograde* atrial activity from a *reentry* SVT — can be forthcoming by obtaining a post-conversion 12-lead ECG. Once sinus rhythm has resumed — this notching in the terminal portion of the QRS should *no longer* be seen.

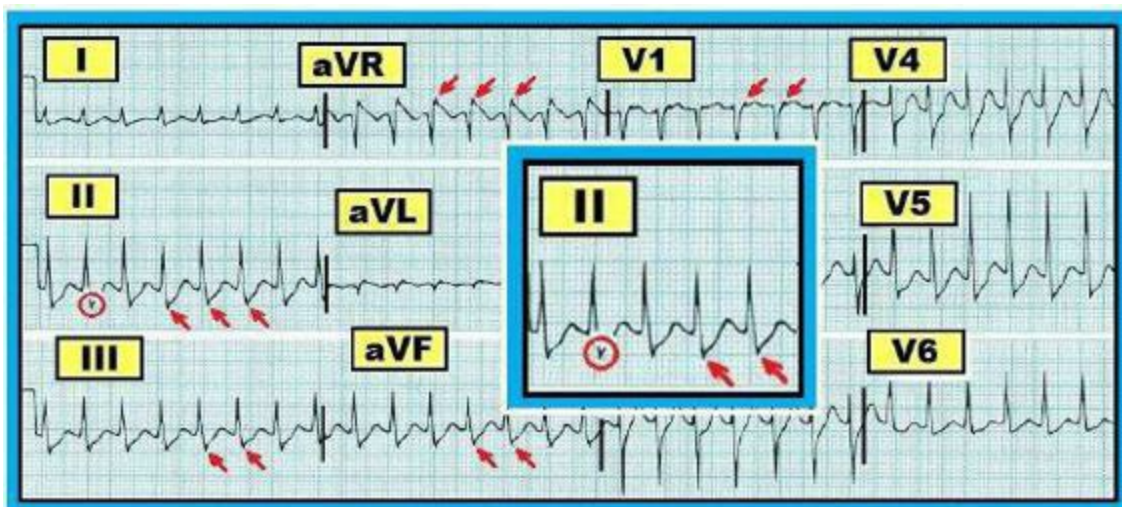


Figure 02.30-1: PSVT. Subtle notching is seen in the *terminal* portion of the QRS in several (but not all) leads (arrows). Identification of such retrograde atrial activity during a regular SVT rhythm indicates a **reentry mechanism** is operative.

02.31 – The “Every-other-Beat” Method (for fast rates)

Accurate determination of heart rate is essential for assessment of the various SVTs (*SupraVentricular Tachycardias*). When the rhythm is regular and the rate is *fast* — calculating rate is most easily accomplished using the “Every-other-Beat” Method (Figure 02.31-1):

- The R-R interval of *every-other-beat* for the PSVT rhythm in **Figure 02.31-1** is precisely 3 large boxes in duration.
- Therefore — **half the rate** will be **100/minute** (300 divided by 3).
- This means that the *actual* rate in **Figure 02.31-1** must be *twice* this amount ($= 100/\text{minute} \times 2 = 200/\text{minute}$).



Figure 02.31-1: PSVT (previously shown in Figure 02.29-1). Heart rate is estimated by the **every-other-beat method**. Since the R-R interval of *every-other-beat* is 3 large boxes in duration — *half* the rate = 100/minute (300/3). The *actual* rate is therefore *twice* this amount = 200/minute.

02.32 – Junctional Rhythms

Junctional (or AV Nodal) rhythms — are regular *supraventricular* rhythms in which atrial activity reflects an AV Nodal site of origin (**Figure 02.32-1**). As opposed to NSR (*Normal Sinus Rhythm*) — in which conduction begins in the **SA Node** and travels downward (*left panel in Figure 02.32-1*) — electrical activity *begins* in the **AV Node** with junctional beats or rhythm (*right panel*) — and is conducted *backward* (ie, *retrograde*) or away from Lead II. As a result, when a **P wave** is seen in **lead II** with junctional beats or junctional rhythm — it will be **negative** (since electrical activity is

traveling away from standard lead II in its path back to the atria).

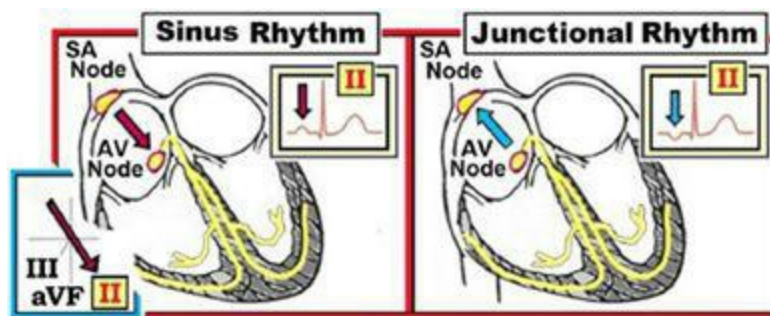


Fig. 02.32-1: Anatomic site of origin for *sinus* rhythm (left) vs AV Nodal beats or AV Nodal (junctional) rhythm (right). As shown in the inserts — the P wave in lead II will be *upright* with sinus rhythm (left insert) but *negative* when a P wave is seen with junctional rhythm (right insert).

02.33 – Junctional Rhythms: P Wave Appearance in Lead II

The 3 possible scenarios for P wave appearance with junctional rhythms is best illustrated by **laddergram** (Figure 02.33-1). *Relative* speed of conduction backward (to depolarize the atria) — compared to *forward* speed of conduction (to depolarize the ventricles) will determine IF the P wave in lead II is:

- **Negative before** the QRS (**Panel B** of Figure 02.33-1) — as would occur if it takes *less* time to conduct back to atria than forward to ventricles.
- **Negative after** the QRS (**Panel D**) — as would occur if it takes *more* time to conduct back to atria.
- **Absent completely** — as would occur IF it takes approximately *equal* time to conduct back to atria as down to ventricles (such that the retrograde P wave is hidden within the QRS — as seen in **Panel C**).

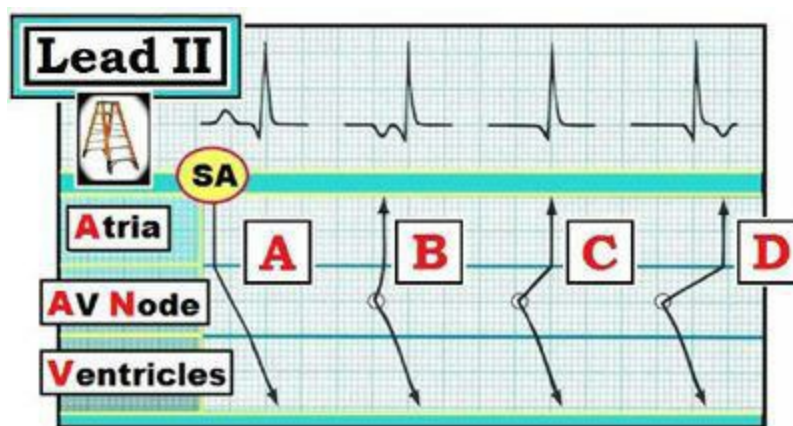


Fig. 02.33-1: Laddergram of the 3 possibilities for P wave appearance in lead II with junctional beats or junctional rhythm. **Panel A** illustrates the **normal forward conduction** of *sinus* rhythm. The impulse originates in the SA Node — travels *through* the atria — slows down a bit as it passes *through* the AV Node — and is then transmitted *down* through the conduction system to the ventricles. **NOTE:** With **junctional beats** (or *junctional rhythm*) — the impulse originates from the **AV Node**. It then travels back (*retrograde*) to the atria and *down* to the ventricles. As a result — the **P wave** in **lead II** may be negative appearing either *before* (**Panel B**) or *after* (**Panel D**) the QRS — or — *no* P

wave at all may be seen (**Panel C**). Clinically — Situation C is *most* common, whereas it is rare to see a P wave after the QRS (*situation D*).

02.34 – Junctional Rhythms: Escape vs Accelerated

There are 3 basic “types” of *junctional* rhythms — with the type determined by the **rate** of the rhythm (**Figure 02.34-1**):

- **AV Nodal Escape Rhythm** — when the junctional rate in an *adult* is between **40-60/minute** (See **Rhythm X** in **Figure 02.34-1**). An AV Nodal “*escape*” rhythm arises because the SA Node is either *delayed* or *fails* in its pacemaking function NOTE: — *This is not necessarily a pathologic rhythm!*
- **Accelerated Junctional Rhythm** — when the junctional rate speeds up to between 61-99/minute (**Tracing Y**) — at which point it *takes over* the pacemaking function from the SA Node.
- **Junctional Tachycardia** — when the rate *exceeds* 100/minute (**Tracing Z**).

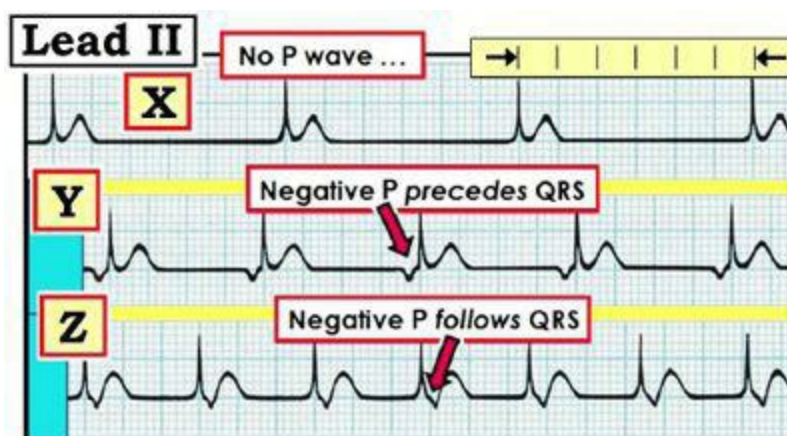


Figure 02.34-1: Junctional rhythms. NOTE: Although we show *no* P wave in X and a *negative* P wave (*before and after the QRS*) in Y, Z — it is *rate* that determines the “type” of rhythm.

Clinical Notes: Junctional Rhythms — The *KEY* to assessing the clinical significance of a junctional rhythm is appreciation of the setting in which it occurs:

- **Junctional “escape”** (40-60/minute) — may be seen on occasion in otherwise *healthy* young adults *without* clinical consequence.
- The same is true in **children** (and *adolescents*) — for whom the normal *escape* rate is a bit faster (50-80/minute).
- In contrast — **accelerated junctional rhythms** (*in which the AV Node speeds up*) — typically occur in a *limited* number of clinical situations (*acute MI; post-operative state; congenital heart disease; shock; digoxin toxicity*). Recognition should prompt concern. The rhythm usually resolves by treating the *underlying* condition.
- **PEARL:** — Regardless of the serum digoxin level — *Think Dig Toxicity* until proven otherwise *whenever* you see an *accelerated* junctional rhythm (*albeit this is less common nowadays given current reduced use of digoxin*).
- Final Note: Clinical implications of **accelerated** vs **junctional tachycardia** are the same (*the*

only difference is whether the rate is over or under 100/minute).

02.35 – Low Atrial vs Junctional Rhythm?

Beyond-the-Core: Technically — a rhythm with a *negative* P wave preceding each QRS complex with *fixed* PR interval could be coming from a **low atrial site** instead of from the AV Node (**Figure 02.35-1**):

- Beats originating from *either* the **AV Node** (Point **X** in **Panel A** of Fig. 02.35-1) or from a **low atrial site** (Point **Y** in **Panel B**) — will *both* be perceived as *moving away* from lead II as they conduct back to depolarize the atria. As a result — a *negative* P wave may precede the QRS complex in *either* case.
- Practically speaking — it does *not* matter whether a beat or rhythm is of junctional or low atrial origin, because clinical implications are *similar* in each case.

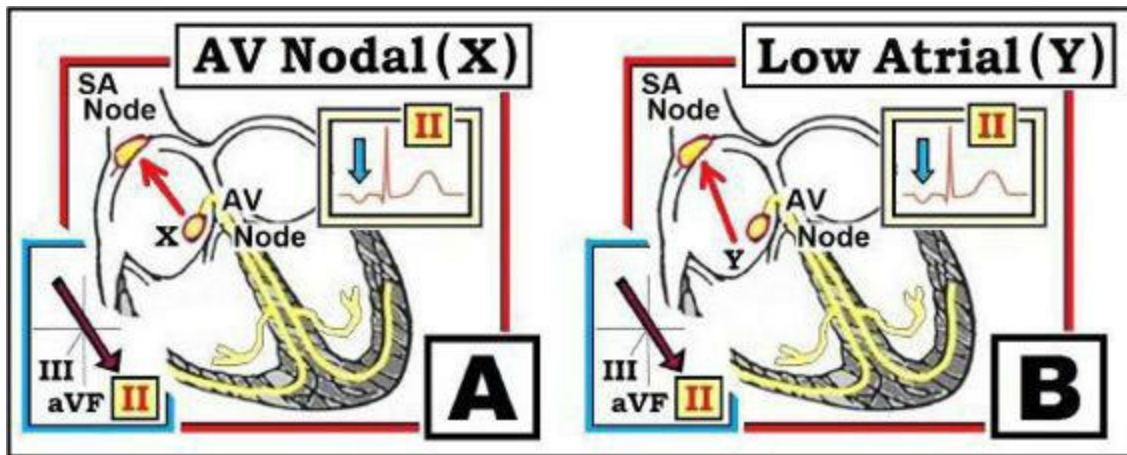


Figure 02.35-1: The presence of a *negative* P wave *before* the QRS may also be seen with **low atrial** as well as junctional beats or rhythm. Atrial depolarization moves *away* from lead II (red arrows) when impulse origin is from *either* the AV node (X) — *or* from a site *low* in the atria (Y).

02.36 – VENTRICULAR (= *wide QRS*) Rhythms

With the exception of the chaotic variability of VFib (*Ventricular Fibrillation*) — the *sustained* ventricular rhythms are most often regular (*or at least fairly regular*) rhythms that originate from a site in the ventricles.

- The **QRS complex** is *wide* — because the *ventricular* origin of these rhythms is *outside* the conduction system. The QRS looks *very* different than sinus beats.
- Ventricular rhythms may arise *either* as “**escape**” rhythms (*if supraventricular pacemakers fail*) — *or* as **usurping rhythms** (*when the ventricular focus accelerates and takes over the pacemaking function from the preexisting supraventricular pacemaker*).
- **P waves** with ventricular rhythms may be: **i) absent**; **ii) unrelated** to the QRS complex; — *or* **iii) retrograde**.

Types of Ventricular Rhythms: — There are 3 basic “types” of *ventricular* rhythms — with the type determined by the *rate* of the rhythm:

- Slow IVR (*IdioVentricular Rhythm*) — Section 02.37.
- AIVR (*Accelerated IdioVentricular Rhythm*) — Section 02.38.
- VT (*Ventricular Tachycardia*) — Section 02.39.

02.37 – Slow *IdioVentricular* Escape Rhythm

The term *slow IVR* is used to describe a regular (*or at least somewhat regular*) ventricular rhythm (*wide QRS; no conducting P waves*) — when the ventricular rate is **between 20-40/minute**. This is the usual rate range of an *intrinsic* ventricular escape focus. As a result — *slow IVR* is often referred to as an *IdioVentricular “escape” Rhythm* (Figure 02.37-1):

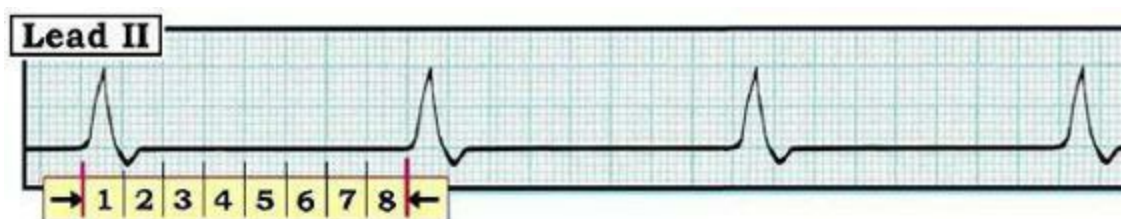


Figure 02.37-1: *Slow IVR*. The ventricular rhythm is regular at a rate of ~38/minute (*the R-R interval = 8 large boxes; 300/8 ~38/minute*). The QRS is wide. No P waves are seen.

- **Clinical Note:** — The occurrence of *slow IVR* is often an *ominous* finding that is commonly seen during the course of cardiac arrest. It may also arise as a ventricular *escape* rhythm in a patient with *complete AV block*.

02.38 – AIVR

AIVR (*Accelerated IdioVentricular Rhythm*) — is said to be present when the rate of the ventricular rhythm is **more than 40/minute** — but does not exceed 110-120/minute (Figure 02.38-1):

- *AIVR* (*also called “slow” VTach*) — is a relatively common *escape* rhythm that may be seen in patients with *acute MI and/or* during reperfusion therapy. It may also occur as a rhythm of cardiac arrest. No treatment is needed IF the patient is asymptomatic (*as this rhythm typically resolves on its own*). Do not give antiarrhythmic drugs (*the drugs may abolish the only escape rhythm the patient has*).



Figure 02.38-1: *AIVR*. The ventricular rhythm is regular at a rate of 75/minute (*300/4*). This is *faster*

than slow *idioventricular* escape — but *slower* than VT. Otherwise — the QRS is wide; there are *no* P waves.

02.39 – Ventricular Tachycardia

VT (*Ventricular Tachycardia*) — is said to be present when the rate of the ventricular rhythm *exceeds* 120-130/minute (Figure 02.39-1):

- By definition — VT is *always* a “**usurping**” rhythm, in that the ventricular focus accelerates and *takes over* pacemaking from the preexisting supraventricular pacemaker. Prompt (*if not immediate*) treatment is essential.

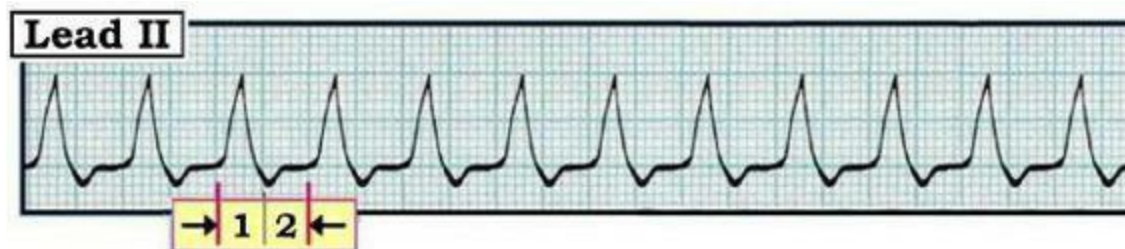


Figure 02.39-1: VT. The ventricular rhythm is regular at a rate of ~150/minute ($300/2$). The QRS is wide; No P waves.

NOTE: — There exists a “**gray zone**” — in which the ventricular rhythm occurs at a rate *between* 110-to130/minute. This falls *in between* the rate range for “*fast*” VT (Section 02.39) and the rate range for “*slow*” VT (Section 02.38).

02.40 – ESCAPE Rhythms: ECG Recognition

IF for *whatever* reason sinus (*or other atrial rhythm*) fails — an **escape rhythm** will hopefully arise from either: i) the AV Node; or ii) the Ventricles (**Figure 02.40-1**).

- The **rate range** for the various “**escape**” pacemakers is easy to remember — because it steps down *sequentially* from the *lower* limit of 60/minute for sinus rhythm (Figure 02.40-1).

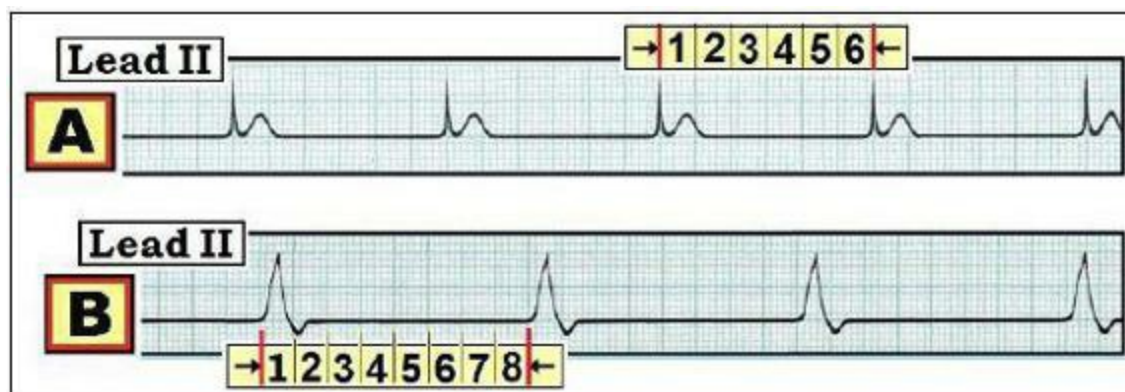


Figure 02.40-1: Escape rhythms. **AV Nodal Escape** (Rhythm **A**) — is recognized by the presence of a narrow QRS rhythm; rate *between* 40-60/minute. **Ventricular Escape** (Rhythm **B**) — is recognized

by the presence of a *wide* QRS rhythm; rate *between* 20-40/minute.

02.41 – PRACTICE TRACINGS: *What is the Rhythm?*



PRACTICE Rhythms:

Interpret the following 5 arrhythmias (*Sections 02.42-thru-02.46*). All rhythm strips are obtained from **Lead II**. The patient was *hemodynamically* stable in each case. *Our answers follow after each tracing.*

- **Hint** — Use the **P's/Q's** and **3R** *systematic* approach for interpreting each rhythm (*Section 02.1*).

02.42 – PRACTICE: *Tracing A*

Interpret the lead II rhythm strip in **Figure 02.42-1**. The patient is hemodynamically stable.



Figure 02.42-1: Practice Tracing A.

ANSWER to Tracing A: The rhythm is slightly irregular, with a rate *between* ~55-65/minute. The QRS complex is *narrow* and is regularly preceded by an *upright* P wave with *constant* PR interval in this lead II monitoring lead. This is **sinus bradycardia** and **arrhythmia** (*Section 02.7*).

- Sinus arrhythmia is a common normal variant rhythm — especially when seen in otherwise healthy children or young adults.
- Sinus arrhythmia is not always benign. Together with sinus bradycardia — it may sometimes be the initial manifestation of SSS (*Sick Sinus Syndrome*). Therefore — *History is everything!* IF the rhythm in Figure 02.42-1 was obtained from an otherwise healthy and asymptomatic young adult — no further follow-up or treatment is indicated.

02.43 – PRACTICE: *Tracing B*

Interpret the lead II rhythm strip in **Figure 02.43-1**. The patient is hemodynamically stable.



Figure 02.43-1: Practice Tracing B.

ANSWER to Tracing B: The rhythm is regular at 75/minute (*the R-R interval is 4 large boxes and $300 \div 4 = 75/\text{minute}$*). The QRS complex is narrow. It is not preceded by any P waves. Thus, this is an **AV Nodal Rhythm**, albeit one that is slightly *accelerated* over the *usual 40-60/minute* rate expected for junctional “escape” in an **adult** (*Sections 02.32-thru-02.34*).

- The fact that no P wave at all is seen in this lead II rhythm strip is abnormal. This means that the impulse is not originating from the SA node.
- It is important to know the **age** of this patient. The normal AV node escape rate in children is between 50-80/minute. On occasion — transient AV nodal escape may be seen in otherwise healthy children. Therefore — this tracing would not necessarily indicate any cardiac pathology IF it was obtained from an otherwise healthy child.
- Always consider the possible **reasons** why a patient may be in an **AV nodal rhythm** — *especially* when the escape rate is slightly accelerated (*as it would be if the rhythm in Fig. 02.43-1 was obtained from an adult*). If the patient is taking Digoxin — Digitalis toxicity is likely regardless of the serum digoxin level. *Other* clinical settings in which an **accelerated junctional rhythm** is likely to be seen include: acute ischemic heart disease — post-operative state — acutely ill patient — congenital heart disease.

02.44 – PRACTICE: Tracing C

Interpret the lead II rhythm strip in **Figure 02.44-1**. The patient is hemodynamically stable.

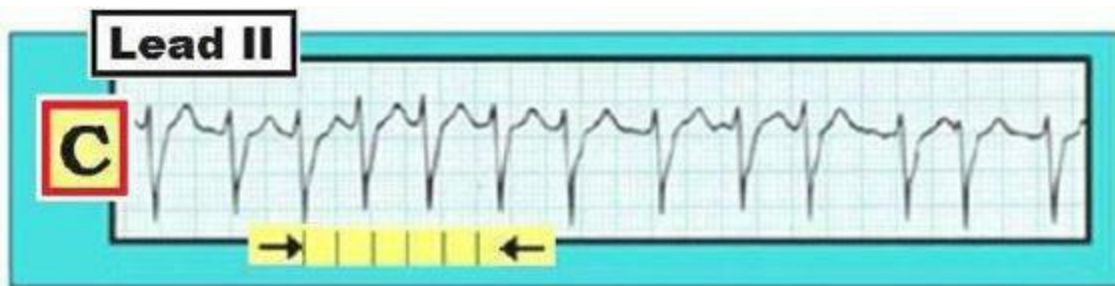


Figure 02.44-1: Practice Tracing C.

ANSWER to Tracing C: The rhythm is rapid and *irregularly* irregular. There are no definite P waves. Although the QRS is wide — this is *not* VT. Instead, the *irregular* irregularity and lack of atrial activity define this rhythm as **AFib**, here with a **rapid ventricular response** (*Section 02.14*). QRS widening is presumably from *preexisting* bundle branch block.

- Note that there are several places in this tracing where it *almost* looks as if there may be P

waves. This is admittedly deceptive. Instead of “P waves” — these are undulations in the baseline that are commonly seen with atrial fibrillation (“*fib*” waves). Gross irregular *irregularity* of the rhythm and *lack* of any repetitive P wave shape define this rhythm as AFib. **Confirmation** could be forthcoming by obtained a **12-lead ECG** with rhythm strip.

- *Beyond-the-Core*: We suspect this patient has LAHB (Left Anterior HemiBlock) — because of the markedly *negative* QRS in this lead II (*Section 07.13*). There may or may not also be underlying RBBB. A 12-lead ECG would be needed to know for sure.

02.45 – PRACTICE: *Tracing D*

Interpret the lead II rhythm strip in **Figure 02.45-1**. The patient is hemodynamically stable.

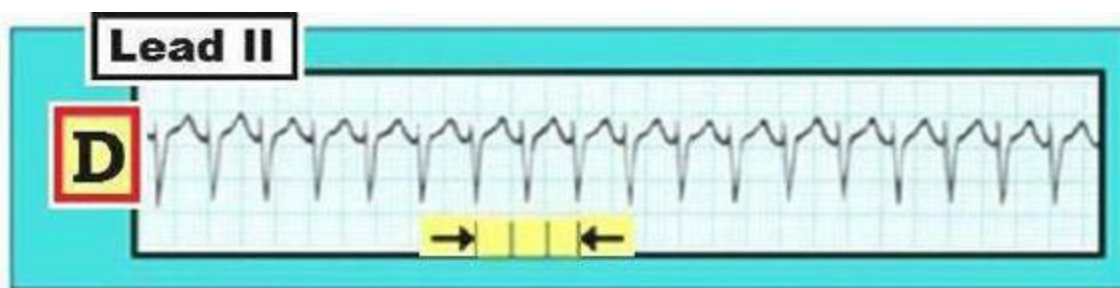


Figure 02.45-1: Practice Tracing D.

ANSWER to Tracing D: The rhythm is rapid and regular. The QRS appears to be narrow. We estimate heart rate by the *every-other-beat* method (*Section 02.31*). The R–R interval of **every-other-beat** (ie, *half* the rate) is about 3 large boxes. This means that *half* the rate is ~100/minute ($300/3$). The **actual rate** is therefore *twice* this, or **~200/minute**. The rhythm is **PSVT** — since a rate of ~200/minute is *too fast* for either sinus tachycardia in an adult or *untreated* AFlutter (See List #2 — *Sections 02.52, 02.53*).

- Although children may manifest sinus tachycardia at rates *over* 200/minute — the *rapidity* of the rate in Fig. 02.45 virtually *excludes* sinus tachycardia in an adult.
- *Beyond-the-Core*: A useful formula to keep in mind is that **maximal heart rate = 220 — age**. This formula is used prior to treadmill testing to estimate what the “target heart rate” should be during the procedure (*the usual goal being to attain at least 85% of maximal predicted heart rate during exercise*). Thus — maximal *estimated* heart rate for a healthy **30-year old** would be $220 - 30 = 190/\text{minute}$. That said — clinical sinus tachycardia in an adult (*who is not performing maximal exercise*) rarely exceeds a rate of 160-170/minute. This is not to say that you will never see sinus tachycardia rates faster than this — but only to say the odds strongly favor an etiology *other than* sinus tachycardia for a *regular* SVT >170/minute.

02.46 – PRACTICE: *Tracing E*

Interpret the lead II rhythm strip in **Figure 02.46-1**. The patient is hemodynamically stable.



Figure 02.46-1: Practice Tracing E.

ANSWER to Tracing E: The rhythm is *irregularly* irregular. The R-R interval appears to vary between 2 and 3 large boxes. The QRS is narrow. Despite the irregularity — this is *not* AFib because P waves are present. P wave morphology *varies* from beat-to-beat. This is **MAT** (Multifocal Atrial Tachycardia — Section 02.16).

- It is important to distinguish MAT from AFib (Section 02.17). *Both rhythms are irregularly irregular.* The difference is that *no* P waves are seen on any of the leads of a 12-lead ECG with AFib. In contrast — *definite* P waves are seen with MAT. As opposed to wandering pacemaker, in which there is gradual transition from one P wave shape to another — with MAT, P wave variation is completely random and varies from one beat to-the-next (*as in Fig. 02.46-1*).
- The **KEY** in management of MAT is to treat the *underlying* disorder. Most often this is *either* pulmonary disease or a combination of *multiple* medical problems including sepsis, shock, acid-base disturbance, etc.


➔ **List #1: *Regular WCT***

NOTE: — We have developed **6 Essential Lists** to facilitate recall of important aspects of 12-Lead/Arrhythmia interpretation. This is the first of our 6 Lists (*Section 00.7*).

The **1st priority** in evaluating any tachycardia is to ensure that the patient is **hemodynamically stable**. IF not — *immediately* cardiovert! If the patient is unstable, it *no longer matters* what the rhythm may be (ie, *VT or SVT with aberrant conduction*) — since the need for *immediate* synchronized cardioversion will be the same.

- But — IF the patient is *STABLE* hemodynamically — an attempt *can* be made to determine the etiology of the tachycardia before proceeding further.
- IF the QRS during tachycardia is **WIDE** — then by definition, the rhythm is a **WCT** (Wide-Complex Tachycardia). Consider the causes = **LIST #1** (Figure 02.47-1).

LIST #1: Common Causes of a Monomorphic Regular WCT of Uncertain Etiology



1. Ventricular Tachycardia (**VT**)
2. VT (esp. IF patient older/ has heart disease)
- Causes #3 thru 8 – VT/VT/VT!!!
9. SVT with *pre-existing* BBB
10. SVT with *aberrant* conduction

Figure 02.47-1: Causes of a **Regular WCT**. Always assume the rhythm is VT until proven otherwise (*See text*).

02.48 – List #1: **KEY Points**

Our reason for putting VT as the first 8 entities in LIST #1 is twofold: **i)** It is *by far* **the most common cause** of a regular (*or almost regular*) WCT in adults when sinus P waves are lacking; and **ii)** It is **the most serious cause!** (Figure 02.48-1).



Figure 02.48-1: Regular *monomorphic* WCT rhythm *without* clear sign of atrial activity. The rate is ~170/minute. Presume VT *until* proven otherwise (*See text*).

We describe the **WCT rhythm** in Figure 02.48-1 as “**monomorphic**” — since QRS morphology *stays the same* during the tachycardia (vs *polymorphic VT* such as *Torsades*, in which QRS

- The **likelihood** that a **WCT** without P waves (as seen in [Figure 02.48-1](#)) is **VT** goes up even more (to at least 90%) — **IF i)** the patient in question is **older** and **ii)** the patient has **underlying heart disease** (prior MI, cardiomyopathy, angina, heart failure). This is true **regardless** of whether the patient is alert — and **regardless** of what the BP might be **during** the tachycardia (**VT can be present even if systolic BP exceeds 180 mmHg**)!
- Availability of a **prior 12-lead ECG** during sinus rhythm may be invaluable for assessing the possibility of **preexisting BBB**. That said — the clinical reality is that you'll often have to initiate treatment **before** knowing for certain what the WCT rhythm is ...

02.49 – Suggested Approach to WCT/Presumed VT

Optimal management of WCT rhythms depends on the *type* of WCT. Assume **VT** until proven otherwise.

- As long as the patient *remains* stable — Trial of *medical* therapy is reasonable. Be ready to cardiovert **IF** at any time the patient *becomes* unstable.
- Get a **12-lead ECG during tachycardia** ([Figure 02.49-1](#)). This will *confirm* that the QRS is *truly* wide — as well as looking for atrial activity in *other* leads. We have found use of **3 Simple Rules** (involving *QRS morphology* and *axis during tachycardia*) may facilitate diagnosis ([Section 02.50](#)).
- Consider *early* use of Adenosine (usually not harmful given its short half-life; may convert SVT and 5-10% of VTs).
- Consider antiarrhythmic (*Amiodarone*; *Procainamide*).
- Be ready to cardiovert if/as needed.

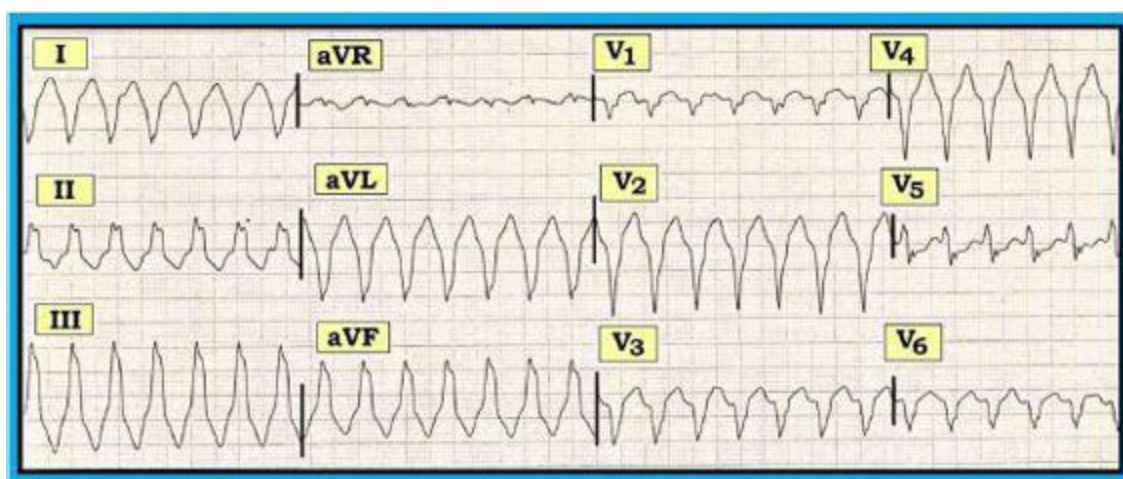


Figure 02.49-1: 12-lead ECG for the rhythm in [Fig. 02.48-1](#). This 12-lead tracing *confirms* that the rhythm is VT (See [Section 02.50](#)).

02.50 – Use of the 3 Simple Rules

We can statistically increase the chance that a *regular* WCT rhythm is VT to over 90% by *applying* **3**

Simple Rules to the 12-lead ECG obtained during tachycardia:

- **Rule #1** — Is there **extreme axis deviation**? The presence of mild or even moderate left or right axis does *not* assist in distinguishing between VT vs SVT. But when there is **extreme axis deviation** (such that the QRS in either lead I or aVF is virtually entirely negative) — then VT is almost certain!
- **Rule #2** — Is **Lead V6 all** (or almost all) **Negative**? IF so — then VT is *highly* likely. Use of this lead V6 criterion is *only* helpful if there is *no more* than a tiny r wave in V6. If the R in V6 is $\geq 3\text{mm}$ — this *doesn't* rule in or out VT.
- **Rule #3** — Is the QRS “**Ugly**”? The “uglier” the QRS complex — the *more* likely the rhythm is VT. The reason for this is that *aberrant* conduction almost always manifests some variation of a conduction defect (RBBB; LBBB; LAHB; LPHB — or some combination thereof). In contrast — VT originates from *outside* the conduction system (and is therefore more likely to be wider and far less organized = “uglier” in its conduction pattern).

Applying the 3 Simple Rules to Figure 02.49-1: If told that the rhythm in Figure 02.49-1 was obtained from an *older* patient *with* underlying heart disease — We'd estimate a **~90% likelihood** of VT based on the finding of a *regular* WCT rhythm *without* sinus P waves (Section 02.47). Use of the 3 Simple Rules allows us to *increase* the **likelihood** of VT to **almost 100%**:

- There is *extreme* axis deviation in Figure 02.49-1 (the QRS complex is completely negative in lead I of this tracing).
- Lead V6 is *almost entirely* negative (at most a tiny r wave is present).
- The QRS in Fig. 02.49-1 is very wide (almost 0.20 second) and formless. We say it is “**ugly**” because QRS morphology does not resemble any form of BBB or hemiblock.

02.51 – FIGURE 02.51-1: 12 Leads are **BETTER** than One

We present the lead II rhythm strip in Figure 02.51-1 to emphasize how **12 leads are better than One**. This rhythm strip was obtained from a patient who was *hemodynamically* stable and tolerating the tachycardia. BP = 120/80 mmHg at the time this tracing was recorded.

- As per the *red* arrow — the rhythm in Figure 02.51-1 was interpreted as sinus tachycardia? *Do you agree?*
- *How would you proceed?*



Figure 02.51-1: Lead II rhythm strip obtained from a patient who was hemodynamically stable. *Is*

this sinus tachycardia?

Answer to Figure 02.51-1: There is a *regular* tachycardia at ~150/minute. Despite the *arrow* suggesting sinus P waves — we are uncertain about QRS width. Since the patient is *hemodynamically stable* — the **KEY** to **management** is to **obtain** a **12-lead ECG** (Figure 02.51-2).

- Some patients in *sustained* VT may *remain* alert and *hemodynamically* stable for *extended* periods of time (*sometimes for hours* — or even longer). Both *short term* and *long term* management will be far more effective IF we can determine with certainty the rhythm diagnosis. Obtaining a **12-lead ECG during tachycardia** will often allow us to do so (**Figure 02.51-2**):

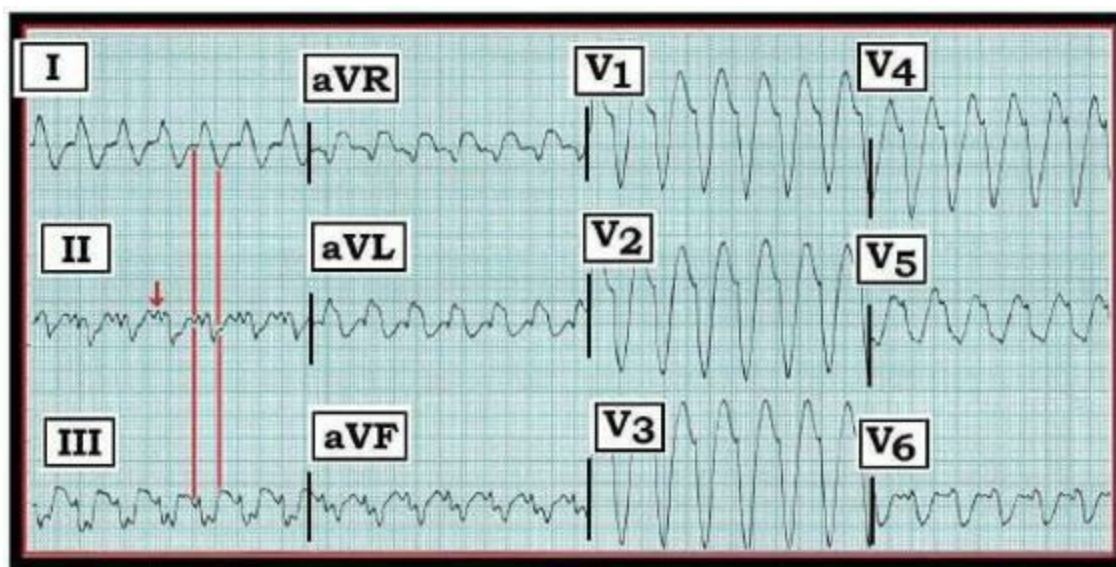


Figure 02.51-2: 12-lead ECG obtained *during* tachycardia. Does the *red arrow* in Figure 02.51-1 represent a sinus P wave or the *initial* part of the QRS complex? (See text).

Interpretation of Figure 02.51-2: It should now be apparent that there are *no* P waves on this tracing. Instead — the red *vertical* timelines show the QRS to be *very* wide. What was initially perceived as a “P wave” in lead II (Figure 02.51-1) — is in fact the initial part of the QRS complex.

- By the **3 Simple Rules** — we can state with **virtual certainty** that the rhythm in Figure 02.51-2 is **VT**! This is because: **i)** there is *extreme* axis deviation (*almost entirely negative QRS in lead aVF*); **ii)** the QRS in lead V6 is *predominantly* negative; and **iii)** the QRS is actually *very* wide and “ugly” (*amorphous, and not resembling any pattern of BBB/hemiblock*).

BOTTOM Line: 12 leads are *better* than one. IF at all uncertain about the etiology of a tachycardia and the patient is *hemodynamically* stable — **Get a 12-lead ECG**. This will often clarify the clinical picture.

- VT may look like a “*narrow tachycardia*” if only a *single* monitoring lead is used. This is because part of the QRS complex may sometimes lie on the baseline. Getting a **12-lead during tachycardia** will confirm QRS duration.


- Some patients may remain alert and *hemodynamically* stable despite being in sustained VT for *extended* periods of time.

➔ **List #2: *Regular SVT***

The 2nd of our 6 Lists also relates to assessment of tachycardia. We again emphasize that IF the patient who is in tachycardia is unstable — *immediately* cardiovert!

- But — IF the patient *is STABLE* hemodynamically — an attempt *can* be made to determine the etiology of the tachycardia *before* proceeding further.
- IF the **QRS** during tachycardia is truly **NARROW** (*in all 12 leads*) — then by definition, the rhythm is an **SVT** (**S**upra**V**entricular **T**achycardia). One of the 3 entities in **LIST #2** (Figure 02.52-1) is then likely to be the cause of ~90-95% of cases of a **Regular SVT**.

LIST #2: Common Causes of a *Regular SVT*
(*without* sign of normal atrial activity)



1. **Sinus Tachycardia** — will rarely exceed a rate of 160-170/minute in an adult. (*Treat the underlying cause of Sinus Tach!*)
2. **Atrial Flutter** — most often has a ventricular rate close to 150/minute.
3. **PSVT (or AVNRT)**.

Figure 02.52-1: Common causes of a **Regular SVT** (See text).

Beyond-the-Core: It is theoretically possible for the QRS complex to be narrow despite *ventricular* origin of the arrhythmia. That is — VT (**V**entricular **T**achycardia) may on rare occasions originate from a site in the Bundle of His or in one of the bundle branches. That said, for practical purposes — IF the QRS is *truly* narrow in *all 12 leads* — the rhythm is supraventricular!

02.53 – The **Regular SVT**: — *Differential Diagnosis?*

The best way to illustrate practical utility of **LIST #2** is through clinical example (**Figure 02.53-1**):

- What is your **differential diagnosis** of the rhythm in Fig. 02.53-1?
- Clinically — *How would you proceed?*



Figure 02.53-1: *Regular SVT at ~200/minute without sign of atrial activity. The patient is stable. What is the rhythm?*

Answer to Figure 02.53-1: The rhythm is regular. The QRS is narrow. By the *every-other-beat* method (Section 02.31) — the R-R interval of *half* the rate is 3 *large* boxes. Therefore — *half* the rate in Fig. 02.53-1 = $300/3 = 100/\text{minute}$. This means that the **actual rate** = $100 \times 2 = 200/\text{minute}$.

- There is *no* clear sign of atrial activity in Fig. 02.53-1. While one *cannot* rule out the possibility that a P wave might be hidden within the ST segment — this is highly unlikely (*would require an extremely long PR interval*).
- The **differential diagnosis** for this *regular* SVT *without* P waves is essentially that shown in LIST #2 (Figure 02.52-1). While there *are* other types of SVT rhythms (ie, *automatic atrial and junctional tachycardias*) — these are *far less common* than the 3 entities we include in List #2. **NOTE:** AFib is *not* a consideration in Figure 02.53-1 — because this rhythm is regular!
- To distinguish between the 3 entities on List #2 — We begin by *looking* at the rate of this *regular* SVT. **IF** the **rate** of a **regular SVT** is **>160-170/minute** — then *both* sinus tachycardia *and* AFlutter become *far less* likely. While **sinus tachycardia** may attain rates of $\geq 200/\text{minute}$ in children — excessively fast rates are unusual in *nonexercising* adults. Therefore, the rate of $\sim 200/\text{minute}$ in Fig. 02.53-1 effectively *rules out* sinus tachycardia in this case.
- **AFlutter** — most often conducts at a ventricular rate that is **close to 150/minute** (*usual range* $\sim 140\text{-}160/\text{min}$). That said — all bets are off regarding the rate range for flutter activity **IF** the patient is on one or more antiarrhythmic drugs (*which may slow the flutter rate*).

BOTTOM Line: By the process of elimination — the *regular* SVT at 200/minute in Figure 02.53-1 is *almost certain* to be PSVT.

- On the other hand — **IF** the rate of this regular SVT would have been *closer* to 150/minute — then *each* of the 3 entities in List #2 would have to be considered.

02.54 – Suggested Treatment Approach for a Regular SVT

Optimal management of the *narrow* tachycardia depends on the *type* of SVT. The “good news” — is that *definitive* diagnosis is *not* essential for appropriate initial management. As long as the patient *remains* stable — there *is* time to consider the following:

- Get a **12-lead ECG** **during** tachycardia. Verify that the QRS is *truly* narrow in *all* leads. You may see sign of atrial activity in *other* leads (*as was evident in Figure 02.19-2 and in Figure 02.30-1*).
- **Fix** any “**Fixables**” you can (*low K⁺/Mg⁺⁺; acidosis; hypoxemia; hypovolemia; ischemia; heart failure, etc.*).
- Consider use of a **vagal maneuver** (Section 02.26).
- Consider empiric use of **Adenosine**. This drug is well tolerated by *most* patients. It will convert *most* PSVT — *and*, it usually produces *transient* slowing of *other* SVT rhythms which may be diagnostic (Figure 02.28-1).
- Consider other AV-blocking drugs (*β -blockers; diltiazem*).

02.55 – FIGURE 02.55-1: Which SVT is present?

Interpret the rhythm for the 12-lead ECG and Lead II rhythm strip that appears in **Figure 02.55-1**:

- What is your *differential* diagnosis?
- What **diagnostic maneuver** might help in diagnosis?

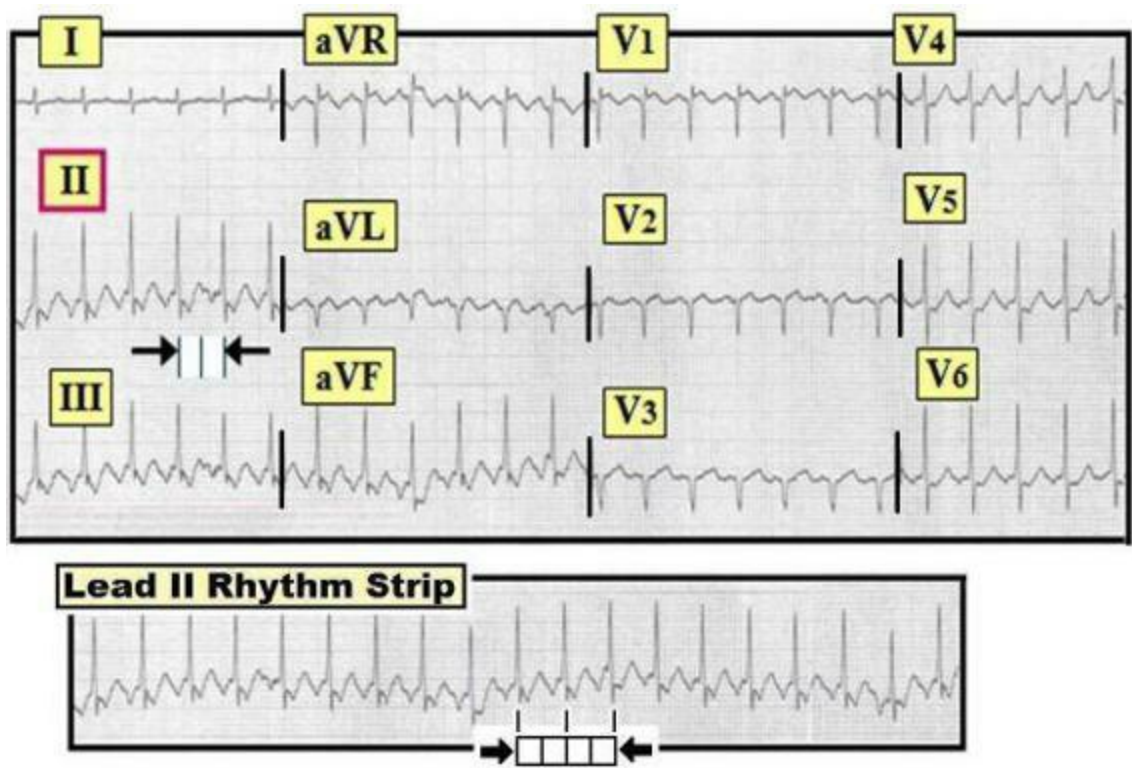


Figure 02.55-1: Regular SVT at ~150/minute. Diagnosis?

Answer to Figure 02.55-1: The rhythm is *regular* at a *ventricular* rate that is *close* to 150/minute (the R-R interval is ~2 large boxes in duration — and $300/2 = 150/\text{min}$). The QRS complex is narrow (ie, *not more than half a large box*). Normal atrial activity is absent — since *upright* P waves are *not* seen in lead II. Instead, there is suggestion of atrial activity having a *negative* deflection in lead II (as well as in other inferior leads). There also appears to be a *negative notching* in the ST segment in each of the inferior leads. *Could this all represent atrial activity?*

- The answer is forthcoming with application of a **vagal maneuver**. The effect of **Carotid Sinus Massage (CSM)** is illustrated in **Figure 02.55-2**. CSM was applied at the moment marked by the **large arrow**.

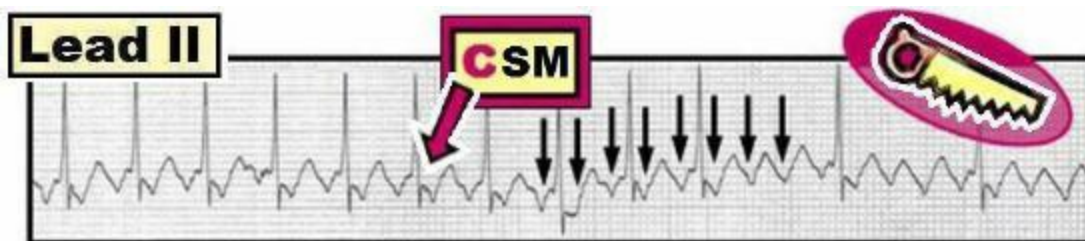


Figure 02.55-2: Application of CSM (*large red arrow*) to the rhythm previously shown in Fig. 02.55-1.

Interpretation of Figure 02.55-2: Application of carotid massage results in *reduction* in the AV conduction ratio from the 2:1 ratio previously seen in Figure 02.55-1. Note slight *lengthening* of the R-R interval following CSM — during which the typical **sawtooth pattern** of **AFlutter** can be readily recognized.

- The **differential diagnosis** of the rhythm in Figure 02.55-1 was that of the causes of a *Regular SVT* = **List #2** (*Section 02.52*). **CSM** in this case **proves** that the rhythm is **AFlutter**.

Clinical NOTE: After **lead II** — the **next best lead** to look for atrial activity in is **lead V1**. This is because *right-sided* lead V1 lies anatomically in close proximity to the atria. Note that a flutter *sawtooth pattern* is suggested in lead V1 on the 12-lead tracing in Figure 02.55-1.

- In retrospect — atrial activity was suggested in *several* leads in Fig. 02.55-1 (*II, III, aVF; aVR, aVL, V1, V2, V5*) — but it was not seen in all leads. CSM *confirmed* the diagnosis.
- **Bottom Line:** The best way to *avoid* overlooking the diagnosis of AFlutter — is to **suspect AFlutter** until proven otherwise *whenever* confronted with a *regular* SVT at a rate *close* to 150/minute but *without* definite sinus P waves (*Section 02.19*).



➔ Premature Beats

Premature Beats — are QRS complexes that interrupt the underlying rhythm by occurring *earlier* than expected. They are **3 basic types** of *early beats* (Figure 02.56-1):

- **PACs (Premature Atrial Contractions)** — when the underlying rhythm is interrupted by an early beat arising from somewhere in the atria *other than* the SA node. As a result — the *early-occurring* P wave manifests a *different* shape (**beat #4 in Fig. 02.56-1**). Most often the impulse is conducted with a *narrow* QRS complex that is *identical* in appearance to that of normal *sinus-conducted* beats. This is because once the premature atrial impulse arrives at the AV Node — its path of conduction through the AV Node and down to the ventricles is the *same* as it is for normally conducted sinus beats.
- **PJCs (Premature Junctional Contractions)** — when the underlying rhythm is interrupted by an early beat arising from the AV Node. Most often the impulse is conducted with a *narrow* QRS that is similar (*if not identical*) to normal sinus-conducted beats. The P wave in lead II is *negative or absent* (**beat #6 in Fig. 02.56-1**; See also Sections 02.32-thru-02.35). Clinically — PJCs are much *less* common than PACs.
- **PVCs (Premature Ventricular Contractions)** — when the underlying rhythm is interrupted by an early beat arising from the *ventricles*. PVCs are *wide* and have a *very* different appearance from sinus beats. PVCs are *not* preceded by a premature P wave (**beat #8 below**). The reason **PVCs** are *wide* — is that the premature impulse arises from a site within the ventricles *away* from the conduction system. It therefore takes *more time* for the premature impulse to be conducted through the ventricles.

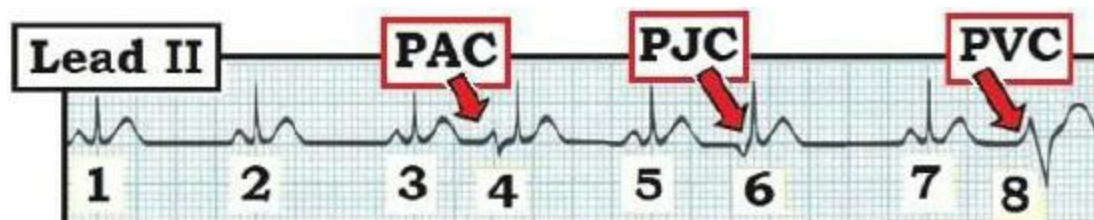


Figure 02.56-1: Sinus rhythm with *premature* beats. These differ from “*escape beats*” that occur late (See text).

Beyond-the-Core: Although **beat #6** in Figure 02.56-1 is labeled as a “PJC” — it could be a *low atrial* PAC instead. There is *NO* way to know for certain (Section 02.35). Management is the same — so clinically, it really does *not* matter whether beat #6 is a PJC vs a PAC arising from a low atrial site (*PACs are much more common than PJCs.*)

- **PEARL** — **Beyond-the-Core:** Although the QRS complex of *junctional* beats is *usually* the same as the QRS of sinus-conducted beats — this is *not* always the case. At times — there may be *slight* variation in QRS morphology for *early-occurring* beats arising from the AV Node.

This is because junctional beats may arise from *different* locations within the AV Node — which may result in a slightly different path of conduction. Thus, the QRS complex will still be narrow — but the R wave may be a little taller or shorter; or the S wave a little bigger or smaller. This is an advanced concept — but it may assist in evaluating occasional complex AV block tracings when it becomes important to determine IF certain beats are being conducted vs representing AV nodal escape.

02.57 – ESCAPE Beats: Timing is Everything ...

It is important to distinguish between **early** (*premature*) **beats** vs **late beats**. Early beats (*PACs*, *PJCs*, *PVCs*) may be problematic — especially if they precipitate runs of tachycardia (**Tracing A** in Figure 02.57-1). In contrast — *late* beats often reflect an “escape” phenomenon which may be beneficial. Were it not for emergence of *ventricular* beats #5, 6 in **Tracing B** (*Fig. 02.57-1*) — the short pause (*between beats #4-5*) may have been much longer.

- Unless accelerated — the **usual rate** of **ventricular escape** is **between 20-40/minute** (*AIVR* was covered in Section 02.38; *Escape Rhythms* in Section 02.40).

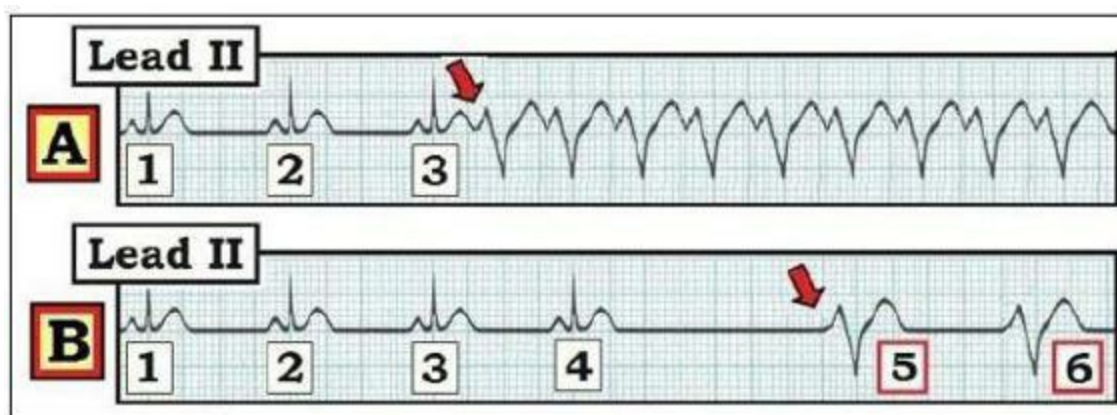


Figure 02.57-1: In **Tracing A** — an *early* beat (*PVC*) precipitates a run of VT after beat #3. In contrast — **beat #5** in **Tracing B** is not a “PVC”, because this beat is not premature. Instead — beat #5 in Tracing B occurs relatively “*late*”, and therefore reflects **ventricular escape** (*See text*).

02.58 – Narrow-Complex Escape Beats

As highlighted by Figure 02.57-1 — **timing** is the *key* parameter for distinguishing between *premature* vs *escape* beats. Consider the events in the 2 tracings shown below in **Figure 02.58-1**:

- What is **beat #5** in both Tracing A and Tracing B of Figure 02.58-1?
- Is the P wave that precedes beat #5 in Tracing B conducting?

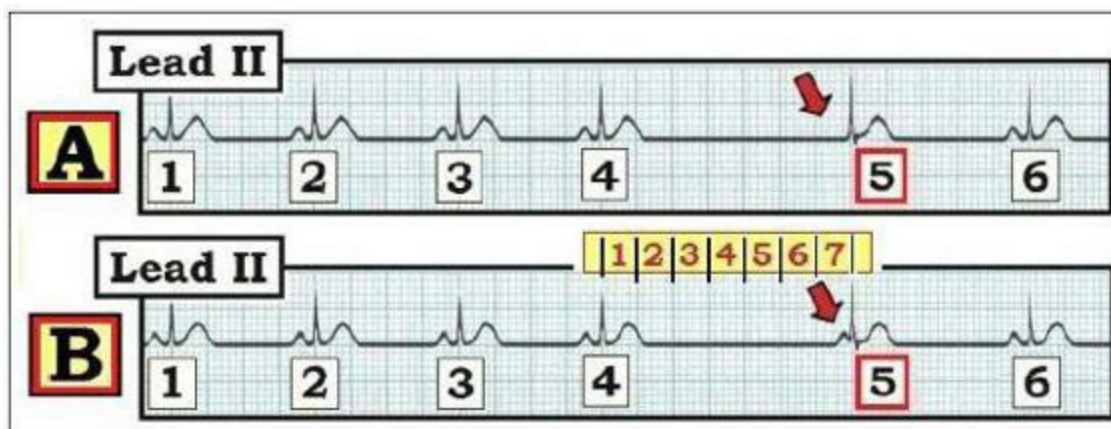


Figure 02.58-1: *Narrow-complex escape beats. Is the P wave that just precedes beat #5 in Tracing B conducting? (See text).*

Answer to Figure 02.58-1: The *underlying* rhythm in Figure 02.58-1 is sinus — as determined by the presence of regular upright P waves preceding each of the first 4 beats with a constant (*and normal*) PR interval.

- **Beat #5** in *both* Tracing A and Tracing B of Figure 02.58-1 is **late**. As a result (*by definition*) — beat #5 can not be either a “PAC” or “PJC”, since it is *not* “premature”.
- Instead — beat #5 is an **“escape” beat**. The occurrence of beat #5 for each tracing in Figure 02.58-1 is a *good* thing! Sinus conduction *fails* after beat #4. If not for the escape beat (*beat #5*) — the pause in *each* case may have been *much* longer...

Focusing first on Tracing A in Figure 02.58-1: The QRS complex of beat #5 is narrow. It is *not* preceded by any P wave. As a result — we know this escape beat *must be* arising from ***within*** the **conduction system**. Possibilities include the AV Node, Bundle of His, or from one of the major fascicles of the bundle branch system. Practically speaking — precise location of the escape site does not matter since initial clinical management will be the same regardless of the site of the escape beat.

- Sinus rhythm resumes in Tracing A after *slight* delay with **beat #6**.
- Beyond-the-Core: QRS morphology of escape beat #5 is similar but not quite identical to QRS morphology of normal sinus beats (*beat #5 is slightly taller than sinus beats — and it has a small narrow s wave that sinus beats don't*). As alluded to in the *PEARL* in *Section 02.56* — recognition of this *slight* difference in QRS morphology for beat #5 (*compared to the QRS of normal sinus beats*) — provides support that *narrow-complex* beat #5 is arising from a supraventricular site *other than* the SA node.

Looking next at Tracing B in Figure 02.58-1: The QRS complex of beat #5 is again narrow. This time beat #5 is preceded by a P wave — albeit the PR interval *preceding* the QRS of beat #5 in Tracing B is far ***too short to conduct***.

- The P wave preceding beat #5 in Tracing B is not a “PAC” — because this P wave is not premature.
- We surmise instead that a brief **sinus pause** (*of 7 large boxes = 1.4 seconds*) ensues after beat

#4. The sinus node finally recovers — but before the P wave preceding beat #5 in Tracing B is able to conduct to the ventricles, an **escape beat** occurs.

- That the **QRS** complex of escape beat #5 in Tracing B is **narrow** tells us the escape focus *must be* arising from a site **within the ventricular conduction system**. Possibilities again include the AV Node, Bundle of His, or one of the major fascicles of the bundle branch system.
- Beyond-the-Core: The R-R interval preceding escape beat #5 in Tracing B is 7 large boxes. This corresponds to an **escape rate** of **~43/minute** ($300/7$). Given that the *normal* AV nodal escape rate range in adults is *between* 40-to-60/minute — this 7-large-box long R-R interval is consistent with the rate of AV nodal escape.
- Normal sinus rhythm resumes in Tracing B after *slight* delay with **beat #6**.

Bottom Line: The complexities of trying to determine IF the site of *narrow-complex* escape beats or rhythms originate from the AV Node, Bundle of His or fascicles of the bundle branch system are of *little* clinical importance! What counts — is that recognition of **late beats** with a **narrow QRS** (as occurs in each tracing of Fig. 02.58-1) — indicates an **escape site** from *somewhere within* the **ventricular conduction system**.

02.59 – PVC Definitions: Repetitive Forms and Runs of VT

Clinically — the occurrence of **repetitive PVCs** (2 or more in a row) — is of much more concern than **isolated PVCs**. In addition to clinical assessment of the patient (ie, *for underlying heart disease; associated medical conditions; electrolyte imbalance; hypoxemia; etc.*) — one aims to assess the *relative* frequency of PVCs — QRS morphology (*uniform or multiform*) — coupling interval — plus the rate and duration of any runs (**Figure 02.59-1**):

- **Tracing A**: Following 3 *sinus* beats — an **isolated PVC** occurs (beat #4). Beats #7,8 — represent a **ventricular couplet**. Note that the *coupling interval* (distance between beat #6 and beat #7) and QRS morphology of this couplet are the *same* as was seen for the *isolated* PVC (beat #4). **Beat #10** — is another PVC. Since its shape is *different*, we say there are **multiform** PVCs in Tracing A.
- **Tracing B**: 3 *sinus* beats — then a **ventricular salvo** (beats #4,5,6). Since the **definition** of “VT” is ≥ 3 PVCs in a row — this is a 3-beat run of VT. **Beat #9** begins a **longer run** of VT. In fact — we have *no idea* of how long this run will last — since **Tracing B** in Figure 02.59-1 is cut off after beat #13. We describe beats #9-thru-13 as a **5-beat run** of **monomorphic VT** at a **rate** of **~150/minute**.

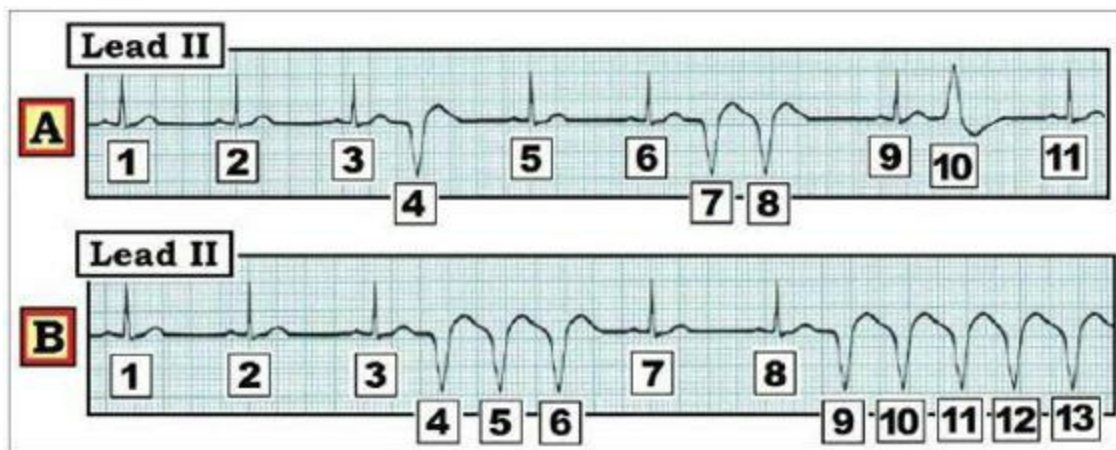


Figure 02.59-1: Various forms of ventricular ectopy (*See text*).

02.60 – Blocked PACs/Aberrant Conduction

Although *most* premature supraventricular beats (PACs or PJC's) are conducted to the ventricles *normally* (ie, with a narrow QRS) — this is not always the case. Instead, PACs (or PJC's) may sometimes occur so early in the cycle as to be "**blocked**" (non-conducted) — because the conduction system is still in the ARP (Absolute Rerefractory Period = 2nd arrow in **Figure 02.60-1**).

- At other times — *early* beats may occur during the RRP (Relative Rerefractory Period) — in which case **aberrant conduction** (with a widened QRS) occurs (3rd arrow).

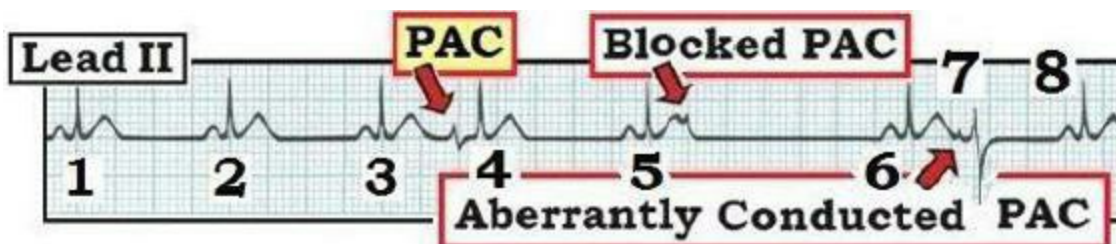


Figure 02.60-1: Blocked vs aberrantly conducted PACs.

NOTE: Whether a PAC will be *blocked vs* conducted normally — or with *aberrancy* depends on *where* in the cycle the PAC occurs (**Figure 02.60-2**). A PAC occurring at point **X** in Fig. 02.60-2 will be blocked (*corresponding to the PAC notching the T wave of beat #5 in Fig. 02.60-1*). In contrast — a PAC that occurs *after* the RRP is over (*point Z* in Fig. 02.60-2) will be conducted normally (*corresponding to beat #4 in Fig. 02.60-1*). It is when the PAC occurs *within* the RRP that *aberrant* conduction is seen (*corresponding to point Y in Fig. 02.60-2 = beat #7 in Fig. 02.60-1*).

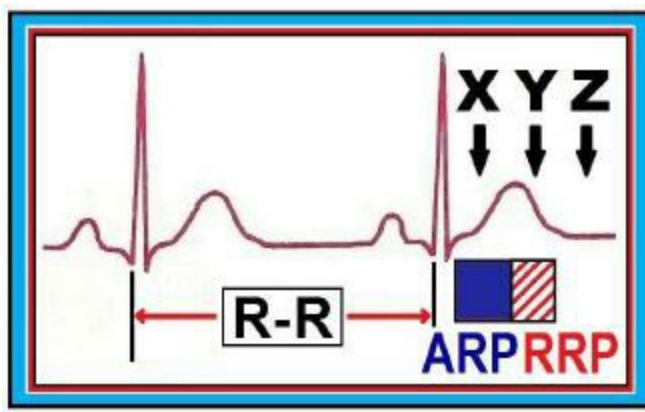


Figure 02.60-2: Whether a PAC (or PJC) will be blocked — conducted normally — or conducted with aberrancy — will depend on *where* in the cycle the beat occurs (See text).

PEARL: The *best* way to diagnose PACs with **aberrant conduction** is when the abnormal beat is clearly *preceded* by a PAC (*beat #7* in Fig. 02.60-1). That said, IF ever in doubt as to whether a widened beat is an *aberrantly* conducted PAC vs a PVC — *Assume* a beat is “**guilty**” (ie, a PVC) until proven otherwise!

- NOTE: **Blocked PACs** may be *subtle* and a challenge to detect. They *will* be found IF looked for — they'll often be hiding (*notching*) the *preceding* T wave (*the PAC that occurs in the T wave of beat #5* in Fig. 02.60-1 — and the PAC marked by the **2nd arrow** in **Figure 02.60-3**). The **KEY** is to recognize *blocked* PACs (and not misdiagnose the pause as a type of AV block). Remember — “**The commonest cause of a pause is a blocked PAC**” (and not AV block)!

Aberrant Conduction: Use of QRS Morphology

Aberrant conduction is most likely to take the form of *some* type of bundle branch block/hemiblock pattern. RBBB aberration is the most common form — because the right bundle branch generally has a *longer* refractory period than other conduction fascicles. That said — any BBB/hemiblock pattern of aberrancy may be seen (RBBB; LBBB; *isolated LAHB or LPHB*; RBBB/LAHB; or RBBB/LPHB). Attention to **QRS morphology** of the *early-occurring* beat may therefore help to distinguish between *aberrant* conduction vs ventricular ectopy.

- The rSR' with taller *right* rabbit ear for **beat #4** in **Figure 02.60-3** — is highly suggestive of **aberrant conduction**.
- The predominantly *negative* QRS deflection for **beat #7** in **Figure 02.60-1** — is suggestive of **LAHB** (Left Anterior HemiBlock) **aberration**.

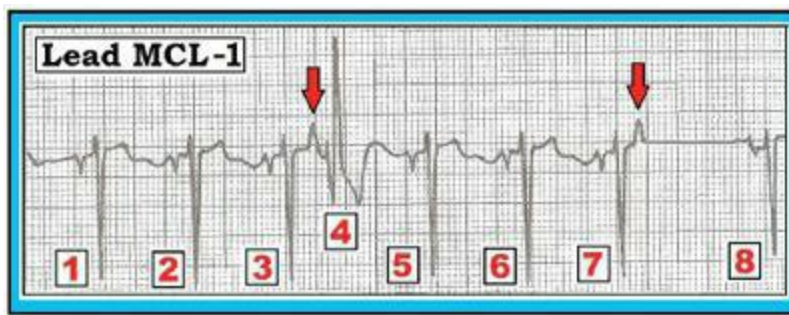


Figure 02.60-3: The 1st PAC in this tracing is *aberrantly* conducted (*1st red arrow that just precedes beat #4*) — but the 2nd PAC is blocked (*2nd red arrow that notches the T wave of beat #7*).

02.61 – PRACTICE TRACINGS – II: *What is the Rhythm?*

PRACTICE Rhythms (II):

Interpret the following 5 arrhythmias (*Sections 02.62-thru-02.66*). All rhythm strips are obtained from **Lead II**. The patient was *hemodynamically* stable in each case. *Our answers follow after each tracing.*

- **Hint** — Use the **P's/Q's** and **3R** *systematic* approach for interpreting each rhythm (*Section 02.1*).

02.62 – PRACTICE: *Tracing F*

Interpret the lead II rhythm strip in **Figure 02.62-1**. The patient is hemodynamically stable.



Figure 02.62-1: Practice Tracing F.

ANSWER to Tracing F: The rhythm is regular at 75/minute. The QRS complex is wide. P waves are present and *upright* in front of *each* QRS complex with a *fixed* PR interval. Thus *despite* QRS widening — this is a ***sinus rhythm***.

- QRS widening is due to ***preexisting BBB*** (*Bundle Branch Block*).
- A 12-lead ECG would be needed to determine the type of BBB that is present.

02.63 – PRACTICE: *Tracing G*

Interpret the lead II rhythm strip in **Figure 02.63-1**. The patient is hemodynamically stable.

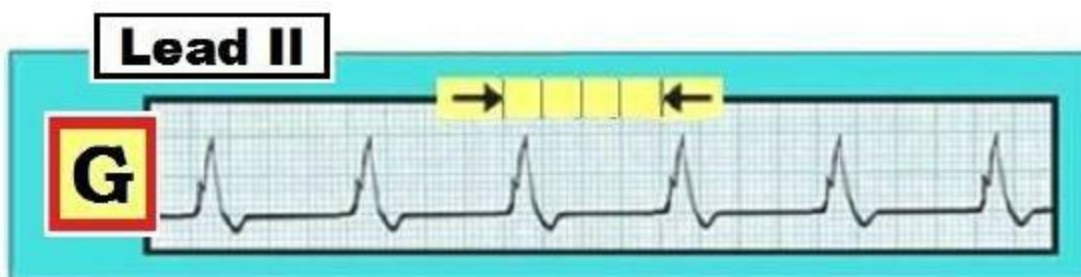


Figure 02.63-1: Practice Tracing G.

ANSWER to Tracing G: The QRS is again wide and regular at 75/minute. However, *no P waves*

are seen in this Lead II. Thus, the rhythm is not sinus. In the *absence* of P waves — QRS widening suggests a **ventricular site** of origin. Because the rate is *faster* than usual for a ventricular escape rhythm — this is **AIVR** (*Accelerated IdioVentricular Rhythm* — Section 02.38).

- The *clinical* setting is key for determining the approach to AIVR. This rhythm is often surprisingly benign when it occurs in the setting of *acute* MI in a *hemodynamically* stable patient. Its occurrence following PCI (*PerCutaneous Intervention*) — may herald *reperfusion* of the *infarct-related* artery. In contrast — prognostic implications are much poorer when AIVR is seen during the course of cardiopulmonary resuscitation.

02.64 – PRACTICE: Tracing H

Interpret the lead II rhythm strip in **Figure 02.64-1**. The patient is hemodynamically stable.

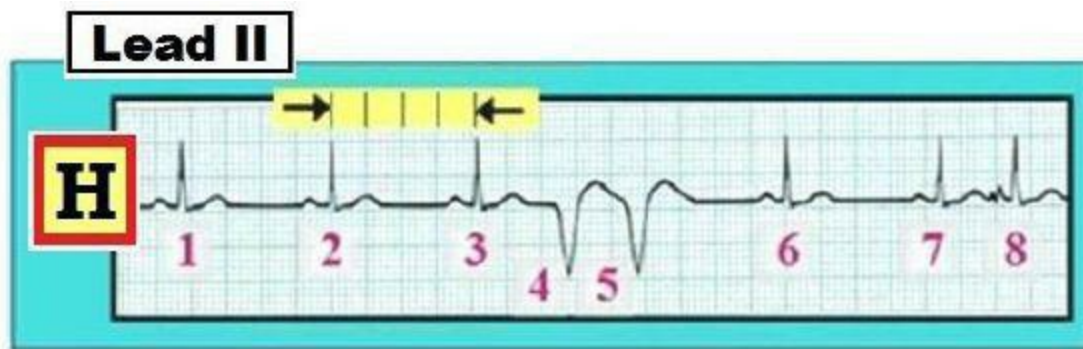


Figure 02.64-1: Practice Tracing H.

ANSWER to Tracing H: The first 3 beats show **NSR** (*Normal Sinus Rhythm*) at a rate of 70-75/minute. Beat #4 occurs early. It is wide, very *different* in morphology from *sinus-conducted* beats, and is not preceded by a premature P wave. **Beat #4** is a **PVC**. Since it is followed by another PVC (*beat #5*) — beats #4,5 form a **ventricular couplet**.

- Beats #6 and 7 are normal *sinus-conducted* beats.
- Beat #8 is early, narrow, of *similar* morphology to normal sinus beats — and is preceded by a different appearing *premature* P wave. Beat #8 is a **PAC** (Section 02.56).
- *Beyond-the-Core:* There appears to be *slight* variation in the R-R interval of normal sinus beats in this tracing (ie, *the R-R interval between beats #1-2; 2-3; and 6-7 is slightly different*). It is difficult to determine IF this is due to underlying *sinus* arrhythmia vs some component of sinus node suppression from the *early-occurring* beats. Clinically — it does *not* really matter whether sinus arrhythmia is present or not, as the primary rhythm abnormality relates to the PAC/PVCs.

02.65 – PRACTICE: Tracing I

Interpret the lead II rhythm strip in **Figure 02.65-1**. The patient is hemodynamically stable.

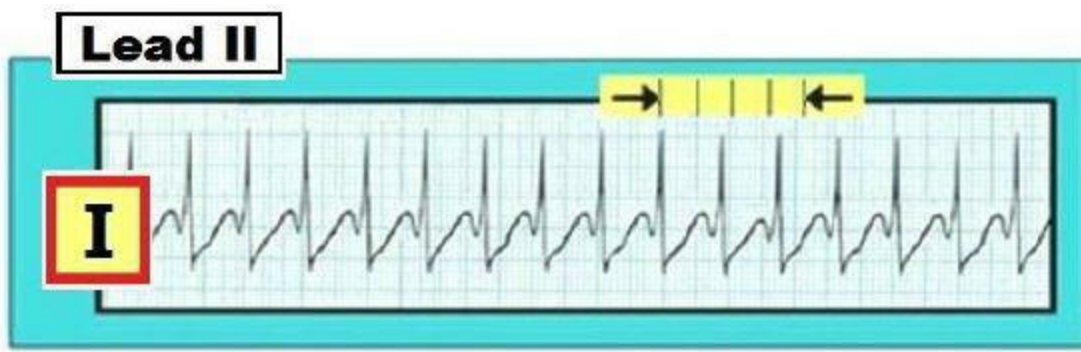


Figure 02.65-1: Practice Tracing I.

ANSWER to Tracing I: The rhythm is rapid and regular at ~180/minute. The QRS appears to be *narrow*, which makes this a **Regular SVT**. It is impossible to determine IF the upright deflection between QRS complexes is a P wave, T wave, or both. The **differential diagnosis** is that of the **3 entities** contained in List #2. These are: **i)** Sinus Tachycardia; **ii)** Atrial Flutter; or **iii)** PSVT (*Section 02.52*). The rapid rate of ~180/minute favors **PSVT** as the most likely diagnosis in this case.

- Sinus Tachycardia *rarely* exceeds a rate of 160-170/minute in a *non-exercising* adult. This is *not* to say that you can never have Sinus Tach at a rate of 180/minute in an adult — but rather to *strongly* suggest you consider *other* diagnostic possibilities as far more likely.
- PSVT most commonly utilizes a **reentry mechanism** involving *some* portion of the AV Node to sustain the tachycardia. As a result — application of a **vagal maneuver** may be *both* diagnostic and therapeutic (*Sections 02.26, 02.27*). If vagal maneuvers are ineffective — administration of an AV nodal blocking drug (*Adenosine; Diltiazem; a β -Blocker*) stands an excellent chance of converting the rhythm.
- Beyond-the-Core: Assuming the patient continues to tolerate the tachycardia — we favor **obtaining** a **12-lead ECG during tachycardia**. Doing so may provide additional clues to etiology — especially when compared to a post-conversion 12-lead tracing (ie, *the thin s-wave notch in the terminal portion of the QRS complex in Fig. 02.65-1 may represent retrograde atrial activity during this reentry tachycardia — similar to notching in the example of PSVT seen previously in Figure 02.30-1*).
- Finally — We note **marked ST depression** in Figure 02.65-1. That said — the amount of ST depression may be exaggerated by a rhythm strip. Never draw conclusions from ST segment deviations (*elevation or depression*) on a rhythm strip. Obtaining a **12-lead ECG** — and repeating the **12-lead after** the tachycardia resolves is the best way to assess likely clinical significance (*if any*) of ST depression seen during tachycardia.

02.66 – PRACTICE: *Tracing J*

Interpret the lead II rhythm strip in Figure 02.66-1. The patient was hemodynamically stable during the *initial* portion of this rhythm strip. Toward the end of the rhythm strip — ***the pulse is lost***.

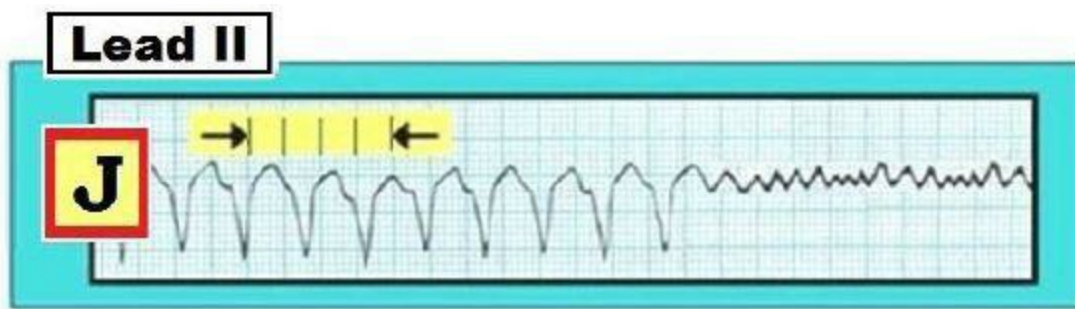


Figure 02.66-1: Practice Tracing J.

ANSWER to Tracing J: The first 9 beats show a **regular WCT** (*Wide-Complex Tachycardia*) at a rate of ~180/minute. *No* P waves are seen. **Presume VT** until *proven* otherwise (= **List #1**, reviewed in Section 02.47). The rhythm then degenerates to **VFib** (*Ventricular Fibrillation*).

- While List #1 *does* allow for the possibility of SVT with *either* preexisting BBB or aberrant conduction — VT should *always* be presumed until *proven* otherwise. Given deterioration of the rhythm to VFib toward the end of the rhythm strip — the first 9 beats are *virtually* certain to be VT.
- Confirmation that the chaotic electrical activity toward the end of the rhythm in Fig. 02.66-1 represents VFib — is forthcoming from the fact that ***the pulse has been lost***. The patient is in need of *immediate* defibrillation!



AV Blocks/AV Dissociation

KEY Clinical Point: Diagnosis of the specific *type* of AV block is far *less* important than the **clinical context** in which the conduction defect occurs. For example — *immediate* treatment is not necessarily needed for all patients with *3rd degree AV block*!

- Clearly — a pacemaker is indicated for *symptomatic* bradycardia that is not readily reversible (*not due to drugs or some other treatable cause*).
- That said, a patient with 3rd degree AV block from acute *inferior* infarction may not necessarily need a pacer — IF the patient is *asymptomatic* with junctional escape at *reasonable* rate and a *normal* BP (*because AV block in this setting may resolve on its own*).

02.68 – Blocked PACs: Much More Common than AV Block

Before describing the AV Blocks — it is well to emphasize the **KEY** clinical concept we introduced in Section 02.60: The **commonest** cause of a **pause** is a **blocked PAC**. IF looked for — **blocked** PACs will be found *much* more often than *any* type of AV block.

- We have already highlighted how to recognize **blocked PACs** by the notching (*or other deformity*) they produce in the T wave at the onset of the pause (*See the T waves of beat #5 in Fig. 02.60-1; and of beat #7 in Fig. 02.60-3*).
- Consider the example in **Figure 02.68-1**. Despite two pauses and the presence of group beating — this rhythm does not represent any form of AV block. *Do you see why not?*



Figure 02.68-1: Despite 2 pauses and group beating — this rhythm does not represent any form of AV block. *Why not?*

Answer to Figure 02.68-1: Keeping in mind that “*the most common cause of a pause is a blocked PAC*” — we routinely look for blocked PACs whenever we see an unanticipated relative *pause* in the cardiac rhythm.

- Note that the T wave of *all* beats in Figure 02.68-1 is smooth and rounded — except for the T waves of the 2 beats at the *onset* of each short pause (= *the T waves of beats #2 and #6*). Instead — the T waves of beats #2 and #6 are both notched! This notching is the result of **blocked PACs** that are *hidden* within these T waves (*arrows in Figure 02.68-2*).

- As we will see momentarily (*in Section 02.72*) — there are *other* reasons why the rhythm in Figure 02.68-2 is *not* 2nd degree AV block of the Wenckebach type: **i)** the atrial rate is *not* regular; **and ii)** the PR interval does *not* progressively increase within groups of beats.
- ***Beyond-the-Core:*** Did you notice that the PR interval preceding beat #7 in Figure 02.68-1 is *shorter* than the PR interval preceding all other beats? This is because **beat #7** is a **junctional escape beat** that occurs *just before* the sinus P wave preceding it has a chance to conduct to the ventricles. We expand on this concept later in this chapter in our discussion on AV dissociation (*in Sections 02.77, 02.78*).

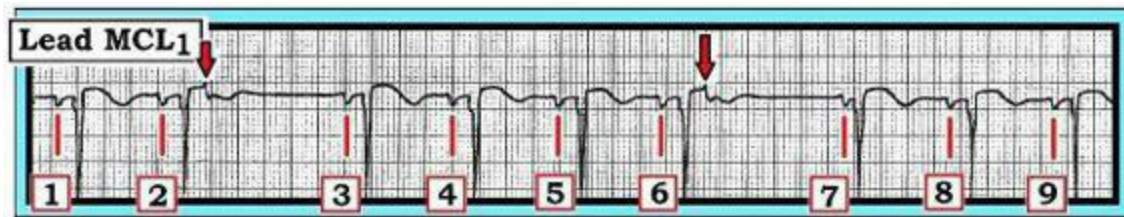


Figure 02.68-2: Sinus rhythm with ***blocked PACs*** (*arrows*). There is *no* AV block in this tracing because: **i)** the P-P interval is *not* regular (*vertical red lines*); **and ii)** the PR interval does *not* progressively increase within groups of beats. *The commonest cause of a pause is a blocked PAC.*

02.69 – The 3 Degrees of AV Block

Diagnosis of the different forms of AV block need *not* be difficult. Simply stated — there are **3 degrees** of AV block:

- **1st degree AV Block** (*Section 02.70*) — in which *all* impulses from above (ie, *from the SA node*) are conducted to the ventricles. It is just that they take *longer* than usual to get there (ie, *more than 0.20 second in an adult*).
- **2nd degree AV Block** (*Section 02.71*) — in which *some* sinus impulses get through the AV node and are conducted to the ventricles, but others do not.
- **3rd degree or complete AV Block** (*Section 02.75*) — in which *no* sinus impulses get through.

NOTE: From a practical, clinical perspective (*as we describe momentarily*) — the diagnosis of *both* 1st degree **and** 3rd degree AV block is surprisingly easy!

- Awareness of this important concept tremendously facilitates diagnosis of the AV blocks. That is — **IF** an AV block is present **and** the block is *neither* 1st degree **nor** 3rd degree — then it **must** be *some form* of 2nd degree AV block.

02.70 – 1st Degree AV Block

First degree AV block is diagnosed by the finding of a ***prolonged PR interval***. This is defined as a PR interval that clearly measures ***more*** than **0.20-0.21 second** = clearly ***more*** than 1 *large* box in duration on ECG grid paper (Figure 02.70-1).

- Practically speaking — **1st degree AV block** is simply a sinus rhythm with a *long* PR interval.
- The ***isolated* finding** of **1st degree AV block** (*even if marked*) is usually not clinically significant (**Figure 02.70-1**). This is especially true when a *prolonged* PR interval is seen in an otherwise *healthy* individual who does *not* have underlying heart disease. In contrast — *new* 1st degree AV block in a patient with *acute* evolving infarction (*especially if associated with other new conduction defects*) — is clearly cause for concern. **Bottom Line:** *Clinical correlation is everything!*



Figure 02.70-1: Sinus rhythm with **1st Degree AV Block**. Each QRS complex is preceded by an *upright* P wave in this lead II rhythm strip with a *fixed* PR interval that clearly *exceeds* 1 large box in duration.

Clinical NOTE: A PR interval that measures **0.20 second** is ***normal***. The PR interval must be clearly more than this amount (ie, >0.21 - 0.22 second) to be “prolonged”.

- **Beyond-the-Core:** Given the overall *benign* nature of *isolated* 1st degree AV block and the clinical reality that otherwise healthy young adults may manifest *slight* PR interval prolongation simply as a result of pronounced vagal tone (*especially in well-conditioned athletic individuals*) — **our preference** is not to call “**1st degree AV block**” until the **PR interval** is **clearly 0.22 second**. We also favor not using the term, “*borderline 1st degree*” when a PR interval in the 0.19-to-0.21 second range is seen in an otherwise healthy individual. All the term “**borderline PR**” means — is that you *almost* have a finding that *even if you had*, would clinically mean *nothing*. Instead — our preference is to describe the **PR interval** as either being “**normal**” or “**long**” (*where long means at least 0.21 second in duration, if not ≥ 0.22 second*).
- **Beyond-the-Core:** **Norms** for the PR interval (*as well as for QRS interval duration*) are ***different in children***. Pediatric hearts are smaller. It therefore takes *less* time for the electrical impulse to travel through the conduction system of a child. For example — a PR interval of 0.18 second would be long for an infant. When in doubt about norms for a *younger-aged* patient — upper limits for the PR and QRS intervals should be *looked up* in an age-specific table.

02.71 – The 3 Types of 2nd Degree AV Block

Second degree AV block is traditionally divided into two types (**Mobitz I** and **Mobitz II**). That said — it is important to recognize that a ***third* type** of 2nd degree AV block also exists (*known as 2:1 AV block*).

- The distinguishing characteristic of the 2nd degree AV blocks — is that some (*but not all*) P waves are conducted to the ventricles (**Figure 02.71-1**).

- **NOTE:** The *atrial rate* should be **regular** (or *almost regular*) when there is AV block (arrows in Figure 02.71-1). It is common to see slight variation (known as **ventriculophasic sinus arrhythmia**) in the setting of 2nd or 3rd degree AV blocks. However — *marked* P wave irregularity — *change* in P wave morphology — or *prolonged* sinus pauses all suggest some phenomenon other than AV block is operative (ie, *blocked PACs*; *escape rhythms without AV block*; *sick sinus syndrome*; *sinus arrest*).

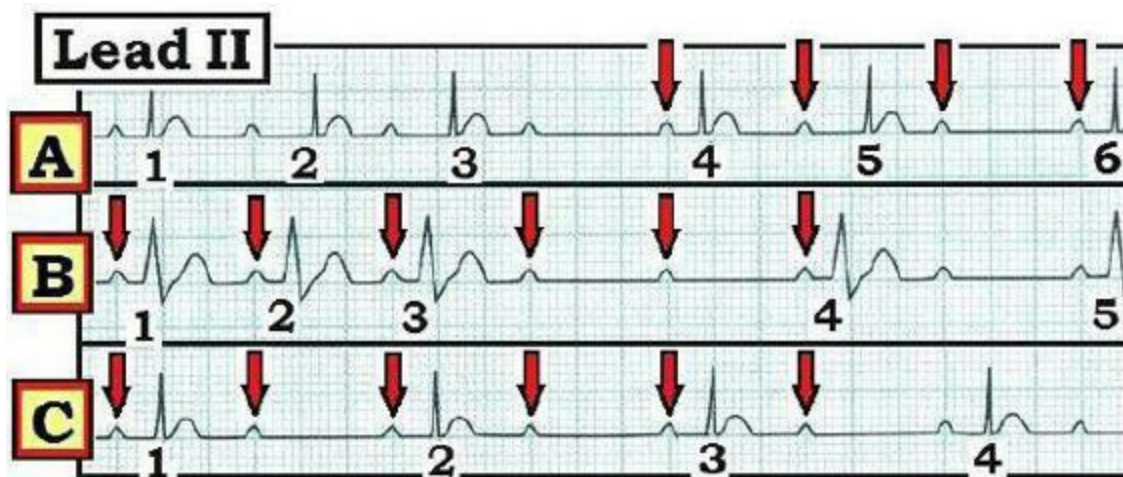


Figure 02.71-1: Sinus rhythm with the 3 types of **2nd Degree AV Block**. In each case — the atrial rhythm is *regular* and some (*but not all*) beats are conducted. **Rhythm A** — Mobitz I 2nd degree AV block with *gradual* prolongation of the PR interval until a P wave is dropped (*Section 02.72*). **Rhythm B** — Mobitz II with QRS widening and a *fixed* PR interval until *sudden* loss of conduction with *successive* nonconducted P waves (*Section 02.73*). **Rhythm C** — 2nd degree AV block with **2:1 AV conduction**. It is impossible to be certain if 2:1 AV block represents Mobitz I or Mobitz II, since we *never* see 2 conducted P waves in a row (*Section 02.74*).

02.72 – Mobitz I 2nd Degree AV Block (= AV Wenckebach)

Mobitz I (*AV Wenckebach*) — is by far the *most* common type of 2nd degree AV block (*accounting for more than 95% of cases in our experience*). **Mobitz I** is **recognized by**: i) Progressive *lengthening* of the PR interval until a beat is dropped; ii) Group beating; iii) A regular (or *at least fairly regular*) atrial rate; and iv) The pause that contains the dropped beat is *less* than twice the shortest R-R interval. These characteristics are known as “*the Footprints of Wenckebach*”. They are *all* present in **Figure 02.72-1**:



Figure 02.72-1: Mobitz I 2nd Degree AV Block (= *AV Wenckebach*). The PR interval *progressively* lengthens until a beat is dropped (*See text*).

Regarding the “*footprints*” of **Wenckebach** that are seen in **Figure 02.72-1**:

- There is “**group beating**”. This takes the form of **2 groups** encompassing beats #1,2,3 and beats #4,5 (*each group being separated by a pause of approximately equal duration between beats #3-4 and between #5-6*).
- The P-P interval is regular (*in this case at a rate of 100/minute*).
- **Within groups** — the **PR interval progressively lengthens** until a beat is dropped. Note the PR interval preceding beat #4 gets longer before beat #5 until the P wave *after* beat #5 is nonconducted. The next cycle then begins as the PR interval shortens prior to beat #6.
- **Beyond-the-Core:** Although AV Wenckebach is usually easy enough to diagnose simply by recognition of *progressive* PR interval lengthening *until* a beat is dropped — awareness of the *other* “footprints” may be helpful confirming the diagnosis in less obvious cases. *Not* all “footprints” are present in each case (ie, *Wenckebach cycles may be atypical*). However, all of the “footprints” are present in Figure 02.72-1. Note that in addition to *group beating* — regular *atrial* rate — and *progressive* PR interval lengthening — that the **pause containing the dropped beat** is *less* than **twice the shortest R-R interval**. Thus, in the first group of beats in Figure 02.72-1 (*beats #1,2,3*) — the *shortest* R-R interval is between beats #2-3. The pause that contains the dropped beat (ie, *the pause between beats #3-4*) measures *less* than twice this R-R interval between beats #2-3.

Clinical NOTE: Second degree AV block, **Mobitz Type I** — usually occurs at the level of the AV node. As a result — the QRS complex with Mobitz I will usually be narrow.

- Mobitz I is most often associated with *inferior* MI — it often *spontaneously* resolves — and it often responds to Atropine (*which works on the AV node*).

02.73 – Mobitz II 2nd Degree AV Block

Mobitz II 2nd degree AV block — is recognized by QRS widening with a **constant PR interval** for **consecutively conducted beats** — until one or more beats are dropped (Figure 02.73-1).

- **Mobitz II** — occurs *lower* down in the conduction system. As a result — the QRS complex is usually wide. The problem with Mobitz II is its disturbing tendency to *abruptly* go from regular conduction of P waves to *nonconduction* of multiple P waves in a row, sometimes leading to ventricular standstill. This explains why **pacing** is almost always needed with *true* Mobitz II 2nd degree AV block.
- The features of Mobitz II are illustrated in Figure 02.73-1. Note that the QRS complex is wide. The underlying atrial rate (*P-P interval*) is regular. Following normal conduction of the first 3 beats on the tracing — there is *nonconduction* of multiple subsequent P waves.
- *Unlike* Mobitz I — the **PR interval** remains **constant** preceding all QRS complexes that do conduct to the ventricles (*beats #1,2,3; #4,5*).
- **PEARL:** The **KEY** for diagnosing Mobitz II is that the PR interval remains constant for *consecutively* conducted P waves (*seen for the PR interval preceding beats #1,2,3 in Figure 02.73-1*).



Figure 02.73-1: Mobitz II 2nd Degree AV Block. The PR interval is *constant* preceding *all* QRS complexes that *do* conduct to the ventricles. Note that the QRS complex is wide and that there are *consecutively* conducted complexes early in the tracing prior to onset of AV block.

02.74 – 2-to-1 AV Block: *Mobitz I* or *Mobitz II*?

The **3rd type** of **2nd degree AV Block** — occurs when there is 2:1 AV conduction — in which *every-other P wave* is conducted (**Tracing C** in **Figure 02.71-1**, which we reproduce below in **Figure 02.74-1**). We can not be certain IF **2:1 AV block** represents *Mobitz I* or *Mobitz II* — because we *never* see 2 beats conducted in a row. Therefore — we simply *don't know* if the PR interval would progressively lengthen IF given a chance to do so.

- NOTE: It is *likely* that the example of **2:1 AV Block** seen in **Figure 02.74-1** represents **Mobitz I** because: **i)** *Mobitz I* is *much more common* than *Mobitz II*; and **ii)** the QRS is narrow. That said — We can not tell for sure (*since we never see 2 conducted beats in a row in Figure 02.74.1*).



Figure 02.74-1: 2nd Degree Block with **2:1 AV Conduction**. Although we *suspect* this is *Mobitz I* (*since the QRS is narrow and Mobitz I is so much more common*) — we can not tell for certain because we *never* see 2 *consecutively* conducted P waves.

Additional CLUES Suggesting Mobitz I: *Serial* tracings and the clinical setting may provide *additional* clues in support of the diagnosis of *Mobitz I* for the rhythm in **Figure 02.74-1**:

- Although possible — it is *rare* for a patient to **go back-and-forth between Mobitz I and Mobitz II** forms of AV block. Therefore, IF there is clear evidence elsewhere that this patient manifested *Mobitz I* (*from the patients chart; from ongoing telemetry monitoring*) — then it is highly likely that the 2:1 AV conduction block seen in **Figure 02.74-1** also represents *Mobitz I*.
- Finally — IF 2:1 AV block occurs in association with **acute inferior infarction** — then **Mobitz I** is far more likely (*since occlusion of the right coronary artery commonly impedes blood supply to both the inferior wall of the left ventricle and the AV nodal artery*). In contrast — acute *anterior* infarction is much more likely to result in *Mobitz II* than *Mobitz I* AV block.

Clinical NOTE: Although there is 2:1 AV block in Figure 02.74-1 — the ventricular rate is *not* overly slow (*the R-R interval is 6 large boxes; the ventricular rate is 50/minute*). As a result — there is an *excellent* chance that this patient may be hemodynamically stable *despite* AV block.

- Immediate treatment may therefore *not* be needed. IF the patient became hypotensive with the rhythm shown in Figure 02.74-1 — initial treatment with Atropine would be reasonable and have reasonable chance to be effective given that the QRS complex is narrow.

FIGURE 02.74-2: Follow-Up Tracing to Figure 02.74-1

A little while *after* the rhythm in Figure 02.74-1 was recorded — the *follow-up tracing* in Figure 02.74-2 is seen on the monitor.

- Does this *new* rhythm labeled **Tracing D** in **Figure 02.74-2** *confirm* our suspicion that the 2:1 AV block seen previously in **Tracing C** (Fig. 02.74-1) is the result of **Mobitz I** 2nd degree AV block?

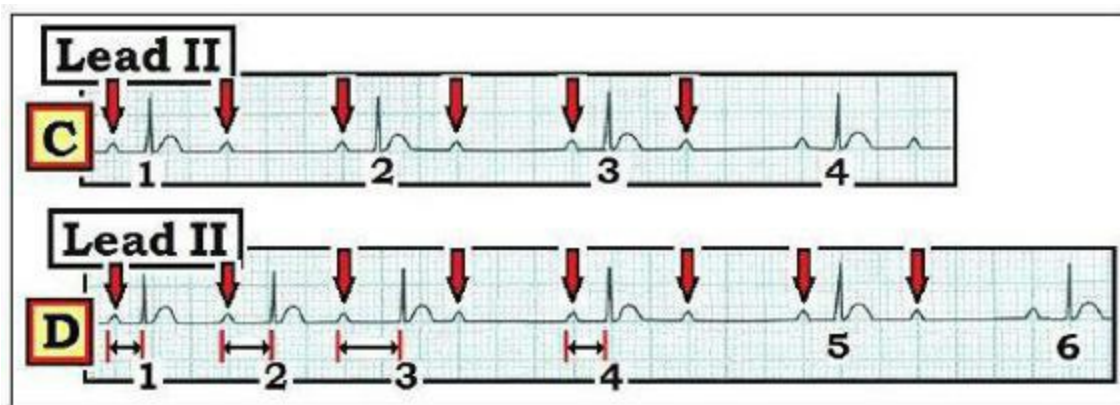


Figure 02.74-2: Tracing D is the *follow-up rhythm strip* obtained a short while *after* Tracing C (Fig. 02.74.1) was recorded. Does tracing D *confirm* that the rhythm in Tracing C represents Mobitz I?

Answer to Figure 02.74-2: The first 3 beats in **Tracing D** show a *definite Wenckebach cycle* (*progressively lengthening PR interval until the P wave after beat #3 is nonconducted*). 2:1 AV block then *resumes* with beat #4 in Tracing D. But because it is *rare* for 2nd degree AV block to alternate between Mobitz I and Mobitz II — it is *virtually* certain that the *entire* sequence of events seen in Tracings C and D of Figure 02.74-2 represent 2nd degree AV block, **Mobitz I**.

02.75 – 3rd Degree (Complete) AV Block

Complete (or 3rd degree) AV block — is said to be present when *none* of the impulses from above (*P waves*) are able to conduct to the ventricles. In this case — there is **complete AV Dissociation** — because *none* of the P waves are related to any of the QRS complexes. The result is that **2 independent rhythms** are going on. One of these rhythms will be *from “above”* (*in the form of regularly occurring sinus P waves*). The *other* ongoing rhythm will be *from “below”* (*in the form of an escape rhythm originating either from the AV node, the His, or the ventricles*). Awareness of

these features facilitates recognition and understanding of diagnostic criteria for what **3rd degree AV block** is — and *what it is not*:

- With complete AV block — *none* of the impulses from above (*sinus P waves*) are able to penetrate the AV node to arrive at the ventricles. As a result — there will be regular (*or almost regular*) atrial activity — and a regular (*or almost regular*) ventricular escape rhythm (**Figure 02.75-1**).
- There is **complete AV dissociation** in **Figure 02.75.1** (ie, *none of the P waves are related to any QRS complexes*). As a result — **P waves** are seen to “**march through**” the **QRS**, occurring at *all* phases of the R-R cycle. This is most easily seen by focusing on *each* QRS complex in *both* Rhythm A and Rhythm B in **Fig. 02.75-1** — and seeing that the PR interval immediately *preceding* each QRS *continually* changes.

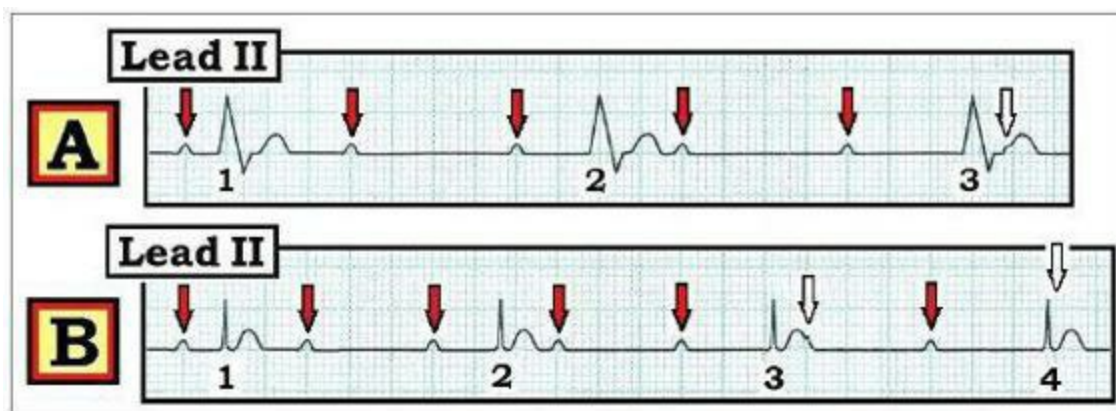


Figure 02.75-1: Anatomic levels of 3rd degree AV block. **Rhythm A:** Complete AV block at the *ventricular* level. There is a regular atrial rhythm (*arrows*) — and the QRS is wide with an *idioventricular escape* rhythm at a rate *between 20-40/minute*. **Rhythm B:** Complete AV block at a *higher* level (probably in the AV node) — as suggested by the presence of a *narrow* QRS escape rhythm at a rate *between 40-60/minute* **NOTE:** *White* arrows in this Figure represent *presumed* P waves that are *hidden* within the ST segment.

NOTE: The *anatomic* level of *complete* AV block may vary. Most commonly — the level of block will occur *below* the AV node with a resultant **ventricular escape** rhythm (**Rhythm A** in **Figure 02.75-1**). When this happens — the **QRS** of the **escape rhythm** will be **wide** and the **escape rate** will be *between 20-40/minute*.

- The anatomic level of block may occur at a **higher** level within the conduction system (*either within the AV node or the Bundle of His*). When this happens — the **QRS** of the **escape rhythm** will be **narrow**. IF the escape rhythm arises from the **AV node** — the **escape rate** will typically be *between 40-60/minute* (as seen in **Rhythm B** of **Fig. 02.75-1**).
- **Beyond-the-Core:** IF the escape rhythm arises from the **Bundle of His** — the QRS complex will again be *narrow*, but the escape rate may be slower. One may therefore surmise the *probable* anatomic level of block based on characteristics of the escape rhythm (*QRS width and escape rate*) — realizing that exceptions may exist in patients with preexisting conduction defects *and/or* influence by medications, hypoxemia, acute ischemia/infarction, etc.

KEY Clinical Point: More important than the anatomic level of block per se in *initial* management is the patient's *hemodynamic* status. For example — a hypotensive patient with altered mental status and *complete* AV block associated with an escape rate of 40/minute will still be in dire need of immediate treatment regardless of whether the escape rhythm manifests a wide or narrow QRS complex.

- *Beyond-the-Core:* Knowing that the escape rhythm QRS complex is *narrow* suggests *greater* potential for reversibility and *beneficial* response to Atropine.

02.76 – PEARLS for Recognizing/Confirming Complete AV Block

Most of the time IF the degree AV block is complete (*3rd degree*) — then **the *ventricular rhythm* should be at least *fairly* regular**. This is because *escape* rhythms arising from the AV node, the His or ventricles are **usually *fairly* regular** rhythms. Exceptions may occur during cardiopulmonary resuscitation — but even then, there will usually be a *recognizable* pattern of ventricular regularity.

- If the ventricular rhythm in a tracing with AV block is *not* regular (or at least *almost* regular) — then it is likely that *some* conduction is occurring (ie, *rather than 3rd degree* — *there is probably high-grade 2nd degree AV block*). Note that the R-R interval is regular for *both* examples of complete AV block shown in Figure 02.75-1.

Confirmation of complete AV block requires that P waves fail to conduct despite being given adequate opportunity to do so. Satisfying this requirement provides the evidence needed to distinguish complete AV block from AV dissociation due to other causes. To do this — one *must* see P waves in *all* phases of the R-R cycle (ie, *having opportunity to conduct* — *but still failing to do so*).

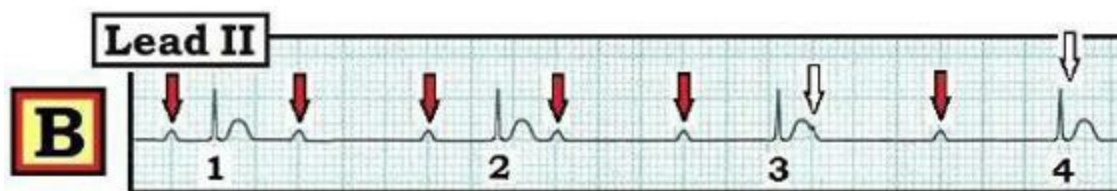
- For example, in Tracing A of Fig. 02.75-1 (which we reproduce below) — the P wave hidden within the T wave of beat #3 (white arrow) would *not* be expected to conduct to the ventricles (because it occurs during the absolute refractory period). In contrast, virtually all other P waves in Tracing A occur at points in the cardiac cycle that should allow opportunity to conduct — yet *none* of them do.



Tracing A from Figure 02.75-1: Complete AV block at the *ventricular* level.

Similarly, in Tracing B of Fig. 02.75-1 (which we reproduce below) — the P wave that notches the terminal portion of the T wave of beat #3 and the P wave hidden *within* the QRS complex of beat #4

each occur at a time when conduction is *not* expected (*white arrows in Tracing B*). However, *all other P waves* in this tracing should have opportunity to conduct — yet *fail* to do so.



Tracing B from Figure 02.75-1: Complete AV block at a *higher level (probably in the AV node)* — as suggested by the presence of a *narrow QRS escape rhythm* at a rate *between 40-60/minute*

KEY Clinical Point: In order to *guarantee* that P waves will have adequate opportunity to conduct — the rate of the escape rhythm should ideally not exceed 50/minute. Escape rates faster than this often result in inopportune timing of P waves that mitigates against having an adequate opportunity to conduct. In such instances (*when the escape rate is well over 50/minute*) — a much *longer* period of monitoring will be needed to ensure that the degree of AV block is complete. Note that the *escape* rate is *below* 50/minute for *both* examples of AV block in *Figure 02.75-1* — further supporting our conclusion that the degree of AV block is indeed complete in *each* case.

02.77 – AV Dissociation

The term, “**AV dissociation**” — simply means that for a certain period of time sinus P waves are *not* related to neighboring QRS complexes. That is, the P waves preceding the QRS are *not* being conducted to the ventricles.

- AV dissociation may be intermittent, recurrent and short-lived – or – it may be persistent and permanent, as occurs with complete AV block.
- **AV dissociation is *never* a “diagnosis”**. Instead — it is a condition caused by “something else”. The task for the clinician is to figure out what the cause of AV dissociation is for any given rhythm. This assignment is far more than academic — since *appropriate* management depends on figuring out the cause of AV dissociation. Active treatment may or may not be indicated. For example — optimal treatment of *complete* AV dissociation with an accelerated junctional rhythm due to digitalis toxicity is simple: IF the patient is hemodynamically stable — simply *stop* digoxin! No pacemaker is needed *despite* that fact that *none* of the P waves on the tracing may be conducting.
- **Complete AV dissociation is *not* the same as complete AV block!** This is one of the most commonly *misunderstood* concepts in all of arrhythmia interpretation. Complete AV block is just *one* of 3 possible causes of AV dissociation. As we will see momentarily — patients with “complete AV dissociation” may actually have *no* degree of AV block at all ...
- Always try to determine *which* of the **3 Causes of AV Dissociation** is operative. The 3 possible causes are: **i) AV block** itself (*either from 2nd or 3rd degree AV block*); **ii) Usurpation** — in which P waves *transiently* do not conduct because an *accelerated* junctional or ventricular rhythm takes over the pacemaking function (ie, “*usurps*” the rhythm); and **iii) Default** — in which a junctional or ventricular *escape* rhythm takes over by “*default*” because the rate of the sinus pacemaker has *slowed down* for whatever reason.

02.78 – FIGURE 02.78-1: *Is there any AV Block?*

Consider the rhythm strip shown in **Figure 02.78-1**. Are the P waves that are seen in this tracing (*red arrows*) being conducted to the ventricles?

- IF so — Which one or two P waves are likely to be conducting?
- Which P waves are clearly *not* conducting?
- Is AV block present? If so — what *degree* of AV block?
- HINT: Do *any* of the P waves that are not conducting have a “chance” to conduct — yet still *fail* to do so?
- Extra Credit: What is the *correct* interpretation of this rhythm?



Figure 02.78-1: Is there AV block? If so — what *degree* of AV block is present? (See text).

Answer to Figure 02.78-1: Full interpretation of the rhythm strip shown here is problematic because: **i)** We are *not* provided with information about the clinical setting; **ii)** The rhythm strip is short (*just over 4 seconds in duration*); and **iii)** We do *not* get to see the *immediately-preceding* rhythm strips that are likely to reveal what is truly going on. That said — what can be stated about the rhythm in **Figure 02.78-1** is the following:

- The **underlying rhythm** appears to be **sinus** — as suggested by **beat #1** which is preceded by an *upright* P wave with *reasonable* PR interval (of 0.18 second) in this lead II monitoring lead.
- The **ventricular rhythm** is **regular** at a rate **just over 50/minute** (the R-R interval is just under 6 large boxes in duration).
- The **QRS** complex is **narrow**. For practical purposes — this means that the rhythm is **supraventricular** (arising from either the SA node as in sinus rhythm — or from the AV node).
- The **P waves preceding beats #3 and #4** in **Fig. 02.78-1** are definitely **not** conducting. They can't be — since the PR interval preceding these beats is clearly *too short* to conduct.
- The P wave preceding **beat #2** is also probably **not** conducting. Although the PR interval preceding beat #2 is *not* necessarily “too short to conduct” — it is clearly *less* than the PR interval preceding beat #1. Given that we know the P waves preceding beats #3 and #4 are not conducting — it is highly likely that the P wave preceding beat #2 is also **not** conducting.
- The **atrial rate** (as defined by the P-P interval) is **regular** at **50/minute** (the P-P interval is precisely 6 large boxes in duration).
- Regardless of whether the P wave preceding beat #2 is or is not conducting — the **“theme”** of the rhythm in **Figure 02.78-1** is that there is **initial sinus bradycardia** (beat #1) at a rate of 50/minute — followed by a period of AV dissociation (since P waves preceding subsequent beats are definitely **not** conducting). This is **AV Dissociation by Default** (ie, default from sinus node slowing that allows emergence of an appropriate junctional escape rhythm at

52/minute).

KEY Clinical Point: There really is *no* evidence of any AV block at all in Figure 02.78-1. *None* of the P waves that fail to conduct have any reasonable chance to conduct (*since the PR interval is simply too short before an appropriate junctional escape rhythm supervenes*).

- What occurs in Figure 02.78-1 is commonly seen among healthy adolescents and young adults during sleep *and/or* undergoing anesthesia. The sinus rate *temporarily* slows — and an *appropriate* junctional escape rhythm arises to the rescue. There is *no* AV block. There is *no* pathology. There is *no* need for acute intervention. Instead — optimal treatment is “benign neglect” (*the clinician is perhaps better off not knowing that transient AV dissociation may be occurring in such otherwise healthy and asymptomatic individuals*).

BOTTOM Line: The *correct diagnosis* for the rhythm in Figure 02.78-1 is: *Sinus bradycardia with AV dissociation by default, resulting in an appropriate junctional escape rhythm at 52/minute*. Nothing more need be written in your interpretation.

- ***Beyond-the-Core:*** Although we assumed that beat #1 in Figure 02.78-1 is conducting — We actually do *not* know for certain that this is true. This is because we *never* see 2 beats in a row that conduct with the same PR interval. Therefore, *it could be* that the patient’s *underlying* rhythm is sinus with *marked* 1st degree AV block — and that beat #1 also manifests AV dissociation. That said, *regardless* of whether or not beat #1 is conducting — the “theme” of this rhythm is still sinus bradycardia with AV dissociation by default. The need for clinical correlation to guide management remains the same (ie, *no immediate intervention is needed IF the patient is asymptomatic and the setting benign*).

02.79 – SUMMARY: Complete AV Block vs AV Dissociation

We summarize the subject of AV Dissociation by Figure 02.79-1 — in which all 3 the causes are illustrated. Note the following:

- **Rhythm A** in Figure 02.79-1: Although there is obvious AV dissociation (*since the PR interval for several P waves is too short to conduct*) — there is *not* necessarily any AV block. We interpret this rhythm as sinus bradycardia with AV dissociation by **default** — resulting in an *appropriate* junctional escape rhythm at 52/minute (See Answer to Fig. 02.78-1).
- **Rhythm B:** There is **AV dissociation by usurpation**. Beats #1 and #2 are sinus conducted with a normal PR interval. The PR interval then shortens. We *know* that P waves preceding beats #4 and #5 in Rhythm B are *not* conducting (*the PR interval is clearly too short to conduct*). No P waves at all are seen after beat #5. The reason is that an **accelerated junctional rhythm** (at 78/minute) has “**usurped**” the pacemaking function from the SA node (*that was beating at 75/minute*). This situation is commonly seen in **digitalis toxicity**. Although the sinus P waves preceding beats #4 and 5 in Rhythm B are *not* conducting (*and the P preceding beat #3 is probably also not conducting*) — *none* of these P waves have any reasonable chance to

conduct. Thus, despite *nonconduction* of P waves — there is *no* evidence of any AV block. It is likely that AV dissociation will spontaneously *resolve* once the cause of the accelerated junctional rhythm is discovered and corrected.

- **Rhythm C:** There is **AV dissociation due to 3rd Degree AV Block**. As opposed to **Rhythm A** and **Rhythm B**, in which *nonconducting* P waves *do not have any reasonable chance* to conduct — P waves in **Rhythm C** occur at *all* points in the cardiac cycle. We would anticipate that virtually *all* of the P waves indicated by *solid red arrows* in **Rhythm C** should have a chance to conduct. Despite this — *none* of these P waves conduct. Instead — P waves “*march through*” the QRS throughout **Rhythm C**. In addition — the ventricular escape rhythm is *less* than 50/minute. **Rhythm C** therefore fulfills criteria for **complete AV block** as the cause of AV dissociation.

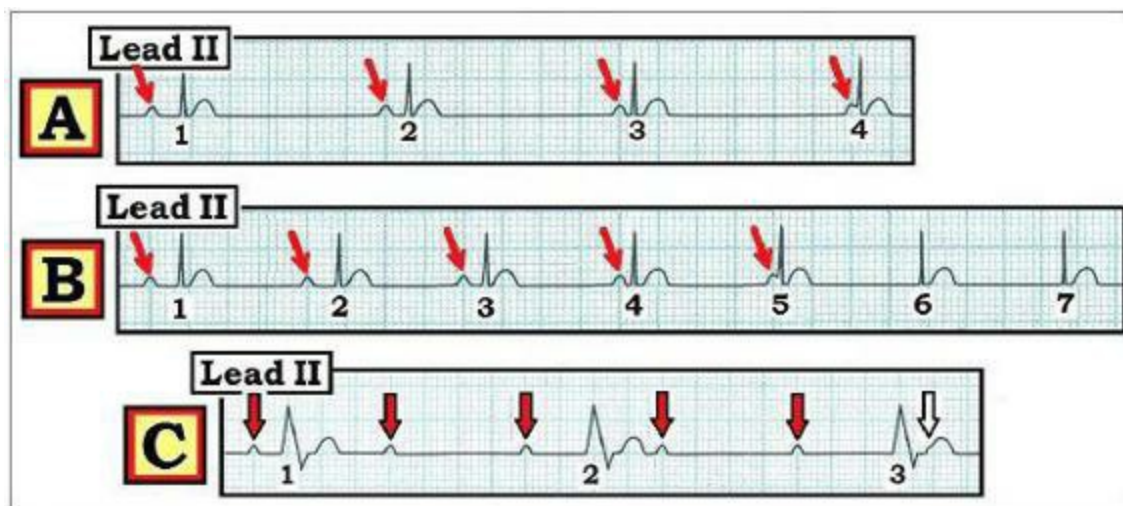


Figure 02.79-1: The 3 causes of AV dissociation. **Rhythm A** — AV dissociation by *default* of the sinus pacemaker which has slowed; **Rhythm B** — AV dissociation by *usurpation* of the rhythm by another *accelerated* pacemaker (*which in this case arises from the AV node*); and **Rhythm C** — AV dissociation due to **AV block** — which could be *either* 2nd degree, or as in Rhythm C, a **3rd degree AV block** (*See text*).

02.80 – High-Grade 2nd-Degree AV Block

A term is needed to describe the situation in **Figure 02.80-1**.

- How would you interpret this rhythm?
- Is there AV block? IF so — Is the conduction disturbance likely to represent Mobitz I or Mobitz II forms of AV block? *Justify* your answer.



Figure 02.80-1: Is the rhythm in this figure Mobitz I or Mobitz II AV block? What is “*high-grade*” AV block?

Answer to Figure 02.80-1: As for assessment of any cardiac arrhythmia — once you have ensured that the patient is hemodynamically stable — “*Watch your Ps, Qs and the 3Rs*” (Section 02.1):

- **P waves** — are present and regular (*red arrows in Figure 02.80-2*).
- **QRS complex** — is narrow (*clearly less than half a large box*).
- **The 3 Rs** — The *atrial* rate is 100/minute; the ventricular rhythm is irregular; *some* P waves conduct — *others do not*.

Impression: *Some* degree of **AV block** is present in Figure 02.80-2. We establish this by recognition of: **i)** a regular *atrial* rate; and ii) the fact that *some* P waves conduct but others *do not*.

- The conduction disturbance in Figure 02.80-2 is clearly not simple 1st degree AV block. We can also recognize *at a glance* that this rhythm is not 3rd degree AV block because: **i)** the ventricular rhythm is *not* at all regular (Section 02.76); and ii) *some* P waves are conducting. Therefore — the rhythm in Figure 02.80-2 must be a form of **2nd degree AV block**.



Figure 02.80-2: Arrows have been *added* to Figure 02.80-1 to indicate *regularly* occurring P waves throughout the tracing. *Some* P waves conduct — but others *don't*. The rhythm is therefore **2nd degree AV block** (See text).

Which Form of 2nd-Degree AV Block? There are features of *each* type of 2nd degree AV block in Figure 02.80-2:

- The first 3 beats are consistent with **Mobitz I** because: **i)** Mobitz I is far more common than Mobitz II; **ii)** the QRS complex is narrow; and iii) the PR interval *progressively* increases from beat #1 — to beat #2 — to beat #3. The P wave after beat #3 is nonconducted. The cycle then resumes with *shortening* of the PR interval prior to beat #4.
- There is **2:1 AV block** for beats #4, 5 and 6 — as *every-other-P* wave is conducted (*confirmed by the constant PR interval preceding the QRS complex of beats 4, 5, 6*).
- Two P waves *in a row* are nonconducted between beat #3 and beat #4. While on occasion, multiple successive *nonconducted* P waves may be seen with Mobitz I 2nd degree AV block — this finding is *much more suggestive* of a more severe **Mobitz II** conduction disturbance (Section 02.73).

Bottom Line: More important than determining the specific “*type*” of 2nd degree AV block for the rhythm in Figure 02.80-2 are: **i)** the *hemodynamic* effect of the conduction defect; and ii) clinical implications and potential need for a pacemaker. As a compromise to reflect our *increased* clinical

concern given **successive nonconduction of P waves** — we favor use of the term “**high-grade**” AV block for the conduction disturbance seen in Figure 02.80-2.

- NOTE: There are *many* variations on the above “theme” — in which the degree of AV block is *not quite complete* and *not* necessarily conforming to either pure Mobitz I or Mobitz II — yet clearly of **increased clinical concern** due to nonconduction of multiple beats with resultant overly *slow* ventricular rate.

02.81 – Ventricular Standstill vs AV Block

Confusion sometimes arises in the terminology used to describe certain ventricular or arrest rhythms related to complete AV block. The 4 rhythms illustrated in Figure 02.81-1 will hopefully clarify the terminology used:

- **Rhythm A** (repeated from Figure 02.75-1) — represents **complete AV block** at the ventricular level. There is a regular *atrial* rhythm (*red arrows*) and a regular *ventricular* rhythm — but *none* of the P waves conduct to the ventricles. Instead — P waves “*march through*” the QRS complex with a *continually* changing PR interval. Note that the ventricular escape rate is slow (*well below 50/minute*) — and that P waves have more than adequate *opportunity* to conduct, yet still *fail* to do so (*Section 02.75*).
- **Rhythm B** — represents **ventricular standstill**. Atrial activity continues (*in the form of regularly-occurring P waves*) — but no QRS complexes are seen. Clinical implications of this rhythm are usually the same as for asystole. Patients with **Mobitz II** 2nd degree AV block sometimes suddenly go from minimal nonconduction of beats to ventricular standstill (*which is why pacing is generally needed for patients with Mobitz II AV block*).
- **Rhythm C** — represents a **slow idioventricular rhythm** — seen here with an escape rate just over 30/minute (*Section 02.37*). This is not a form of AV block — because there are *no* P waves. Instead — following sinus arrest and failure of an AV nodal escape pacemaker to arise — there *fortunately* is appearance of a **ventricular escape rhythm** (*without which there would have been asystole*).
- **Rhythm D** — represents **asystole**. There is no sign of any electrical activity (*No P waves; No QRS complexes*).

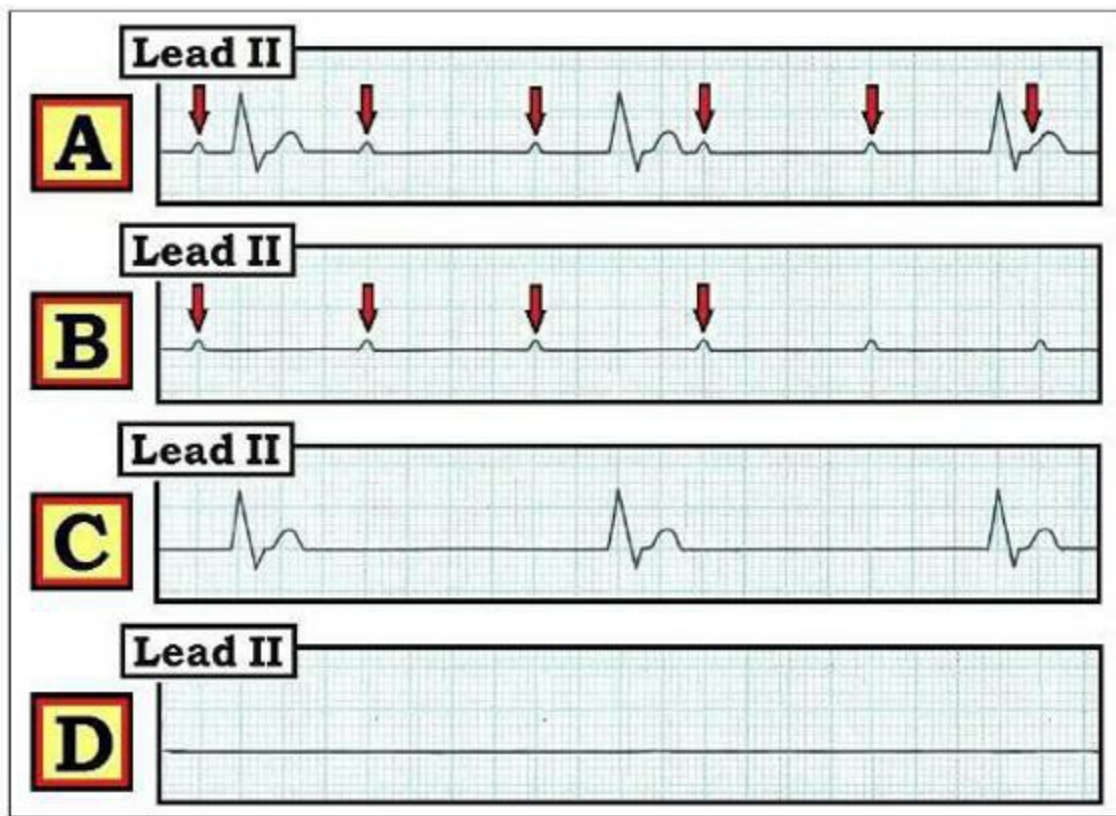


Figure 02.81-1: Clarification of *complete* AV block (**Rhythm A**) — *ventricular standstill* (**Rhythm B**) — *slow idioventricular escape* (**Rhythm C**) — *and asystole* in **Rhythm D** (See text).

02.82 – Hyperkalemia vs AV Block

The incidence of **Hyperkalemia** has clearly increased in recent years. Reasons for this increase are multiple — but include the obesity epidemic with corresponding increase in the incidence of diabetes and improved treatment of diabetes resulting in increased longevity. As a result — many more patients with diabetes now live long enough to develop complications associated with *end-stage* kidney disease. Hyperkalemia is perhaps the most immediately *life-threatening* complication seen in this group of patients.

- Many more patients than ever before are now on **dialysis**. Hyperkalemia should always be thought of in such patients whenever they present with either an unusual (*or bizarre*) 12-lead ECG *and/or* a cardiac arrhythmia.
- Chronic kidney disease patients who are not yet on dialysis may also develop hyperkalemia — especially if any of a number of **predisposing situations** are present. Among others, these include: **i)** taking potassium-retaining medications (*ACE-inhibitors; angiotensin-receptor-blocking drugs; certain diuretics; potassium supplements*); **ii)** dehydration; **iii)** acidosis; **iv)** abrupt reduction in urine output; and **v)** cardiac arrest.
- **Hyperkalemia** is **notorious** for its ability to mimic other ECG conditions (*Section 11*). Among ECG changes produced by more severe degrees of hyperkalemia (*common once serum potassium exceeds 6-6.5 mEq/L*) include: **i)** marked **peaking** of **T waves** in multiple leads; **ii)** **QRS widening**; **iii)** **reduced P wave amplitude** — sometimes with complete **loss** of **P waves** (*despite persistence of sinus conduction*); **iv)** bizarre frontal plane **axis shifts**; and **v)** **ST-T wave changes** that may mimic ischemia/acute infarction (*ST elevation or depression; T wave peaking and/or deep T wave inversion*).

Relevance of the above information on Hyperkalemia to this Section on AV Block is highlighted by the rhythm in **Figure 02.82-1**.

- Should the patient whose rhythm is shown in **Figure 02.82-1** be treated with Atropine *and/or* pacing?
- **HINT:** How might your answer to this question *change* **IF** told that this patient was alert and had a history of chronic kidney disease?



Figure 02.82-1: Ventricular rhythm at ~40/minute. How should this patient be treated? (See text).

Answer to Figure 02.82-1: A regular ventricular (*wide QRS*) rhythm is seen in this tracing at a rate of ~40/minute. There are no P waves. While our *initial* impression is that of a **ventricular escape rhythm** (*as most commonly occurs in a setting of cardiac arrest*) — pending the need for *additional* information, ***other possibilities*** should be entertained. For example — **IF** told that this patient was hemodynamically stable, alert and on longterm dialysis (*or that he/she had a history of chronic kidney disease*) — then a ***stat potassium*** value becomes essential for optimal management:

- QRS *widening* — the *slow* ventricular rate — *lack* of P waves — and suggestion of T wave *peaking* in **Figure 02.82-1** are *all* consistent with a *possible* diagnosis of hyperkalemia.
- **IF** the diagnosis is **hyperkalemia** — then ***immediate treatment*** would be *very different* than treatment of slow ventricular escape in a patient with cardiac arrest whose serum potassium is normal. As opposed to Atropine, use of a pressor agent *and/or* Pacing — **IV Calcium, Bicarb and/or D50 plus Insulin** may become the treatment(s) of choice.

02.83 – FIGURE 02.83-1: *Is there any AV Block at all?*

- We conclude this brief section on AV block with the rhythm in **Figure 02-63**. Is there AV block? If so — What *type* of AV block is present?



Figure 02.83-1: Is there AV block? If so — What type? (See text).

Answer to Figure 02.83-1: Although it is tempting to interpret the rhythm in this tracing as 2:1 AV block — this is not what is happening!

- Assuming the patient is *hemodynamically* stable — We once again assess the 5 key parameters for rhythm determination by the *systematic Ps, Qs and 3R Approach*: The QRS complex is narrow. P waves are present — and 2 P waves are seen for each QRS (*blue and red arrows in Figure 02.83-2*) — but P waves are not regular in this rhythm strip. This is the 1st clue that the rhythm in Figure 02.83-2 does not represent a form of AV block (*Section 02.71*).



Figure 02.83-2: Red and blue arrows have been added to Figure 02.83-1 (*See text*).

- Every-other-P wave* in Figure 02.83-2 is conducting. We know this — because the PR interval preceding *each* QRS on the tracing is constant (*blue arrows*). Therefore — the ***underlying rhythm*** is **sinus**.
- Every-other-P wave* is early (*red arrows*). Note P wave morphology of each *early* P wave is different in shape (*triphasic*) than the *biphasic* P wave of sinus beats. Thus, the rhythm is **atrial bigeminy**. *Every-other-P wave* is a PAC that occurs *so early* that it is blocked (*red arrows*).
- Bottom Line:** The rhythm in Figure 02.83-2 is a fitting way to end this Section on AV Blocks. *The most common cause of a pause* — is a **blocked PAC**. Blocked PACs are far *more* common than any form of AV block. They will be found IF looked for. Remembering this clinical reality (*Section 02.68*) — and remembering to *always* assess *each* cardiac rhythm ***systematically*** by the **Ps, Qs & 3R Approach** will facilitate recognizing AV block when it *does* occur — and help *avoid* misdiagnosis when instead one of the *AV-block-mimics* is present.

Doing an ECG / Errors

The importance of correct lead placement and awareness of potential technical mishaps cannot be overstated. We begin this Section with overview comments on lead derivation and placement. This will enhance appreciation of the PEARLS we then present on *recognizing artifact and* lead placement errors.

03.1 – Limb Leads: Basic Concepts/Placement

A *standard* ECG is recorded by use of **10 electrodes**, which results in a recording that shows **12 ECG leads**. Four of these electrodes are “limb lead electrodes” — which are placed on each of the 4 extremities (**Figure 03.1-1**). The other 6 electrodes are placed on the chest, as described in Section 03.6.

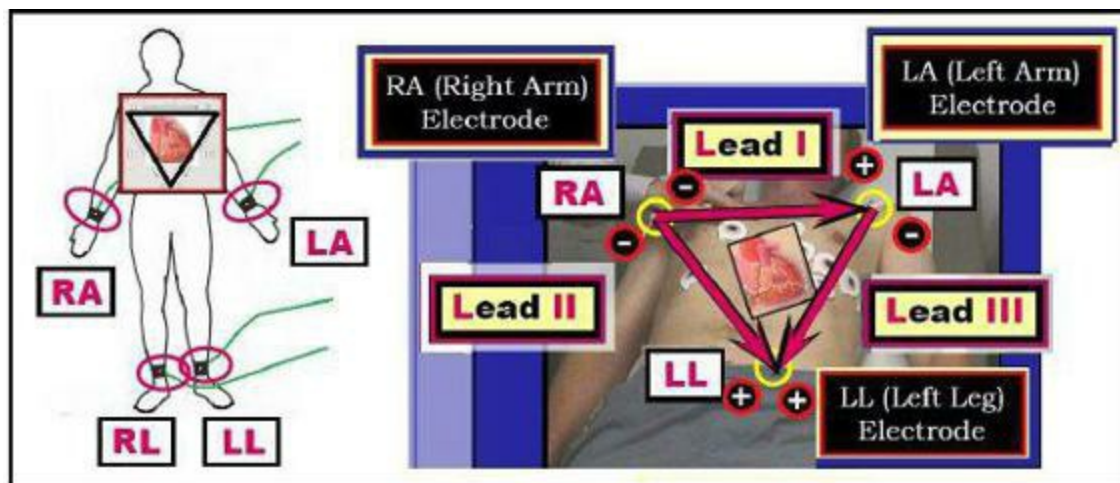


Figure 03.1-1: Limb lead electrodes are placed on each extremity (*left panel*). Derivation of the 3 *standard* limb leads (*leads I,II,III*) is shown in the *right panel*. **NOTE:** The **4th extremity electrode** is placed on the **right leg (RL)**. This RL electrode had traditionally been used as a ground lead — though in modern ECG recording systems, it is now used more to reduce external interference.

Note on the *left* in Figure 03.1-1 — that the **4 limb lead electrodes** are shown placed on the 4 extremities — but that on the *right* we schematically suggest a more *proximal* location for limb lead electrode placement.

- Depending on institution practice where you reside — you are likely to encounter *other* slight variations in such placement. The “good news” — is that since (*for the most part*) — the body is a fairly homogeneous conductor of electricity — there will usually be no more than minimal (*if any*) variation in QRS morphology as a result of such variation in electrode lead placement.
- That said — Realize that there may on occasion be variation in QRST morphology because of different limb lead electrode placement (*Kligfield et al. — AHA/ACC Recs. JACC 49:1109-1127, 2007*). For example — *small Q* waves may be present on one tracing and not on another. This could potentially lead to misinterpretation of new or prior infarction. **PEARL:** *Be consistent!* Do not change electrode placement at your site of practice from one day-to-the-next.

Ensuring that all who record ECGs in your site of practice use the *same* electrode lead placement procedure will lead to consistency — and enable effective use of comparison serial tracings to establish that new findings are truly "new" (*and not the result of different recording technique*).

- Despite one's best efforts to be consistent in recording limb lead placement — there may still on occasion be instances when you'll need to deviate slightly from procedure. Examples include patients with limb amputation *and/or* patients with significant tremor that impedes more distal placement. The **KEY** in such instances is to note *on the actual ECG* what the problem is (ie, *marked tremor*) — and what the change in recording procedure is that you have made (ie, "*used more proximal lead placement*", or *other adjustment*). This way — **IF** you encounter a change in QRST morphology — you'll know that altered technique may have had a contributing role in producing that change.

03.2 – Why 10 Electrodes but 12 Leads?

The reason there are only 10 electrodes (*but 12 standard ECG leads*) — is that **one** of the electrodes (*on the right leg*) is *not* used to derive ECG waveforms — and the 6 limb leads are derived from the remaining **3** extremity electrodes (*on the right arm, left arm, and left leg*). Together with the **6** chest lead electrodes — this makes a total of $1+3+6 = \mathbf{10}$ electrodes used to record **12 ECG leads**.

- The theoretical basis of electrocardiography arose from the premise put forth by **Willem Einthoven**, who in 1903 invented the first ECG machine. Einthoven stated that the **heart** lies in the **center** of an **equilateral electrical triangle** (Figure 3.2-1) While anatomically, the heart is *not quite* at the "center" of this triangle — Einthoven's original premise remains accurate enough that more than 100 years later, this concept still explains many of the fundamental principles of electrocardiography.
- An ECG recording is simply the **graphic representation** of the **heart's electrical activity**. It is *nothing more* — and *nothing less*. This electrical activity is recorded by the 10 monitoring electrodes — and then displayed in a series of waveforms that make up the standard 12-lead ECG.
- **NOTE-1:** An electrical wavefront travels from a *negative* to *positive* recording electrode. A wave of depolarization that is seen as *approaching* a monitoring electrode — will write an **upward (positive) deflection** on ECG in the lead it approaches. It will write a **downward (negative) deflection** in the lead it moves away from.
- **NOTE-2:** Einthoven *arbitrarily* selected the **left shoulder** (= **LA** — *or left arm*) to be **positive** when recording **lead I**. The groin (= **LL** — *or left leg*) electrode was selected to be positive when recording leads II and III. In this way — a *majority* of QRS complexes in the limb leads will be predominantly positive in *most* leads in the electrocardiograms of *most* individuals (Figure 03.2-1).
- **NOTE-3:** As will be discussed in Section 03.3 — the ECG waveform that is seen on ECG for the 3 *standard* limb leads (*leads I,II,III*) is derived from the *difference* in electrical potential *between 2* of the limb lead electrodes.

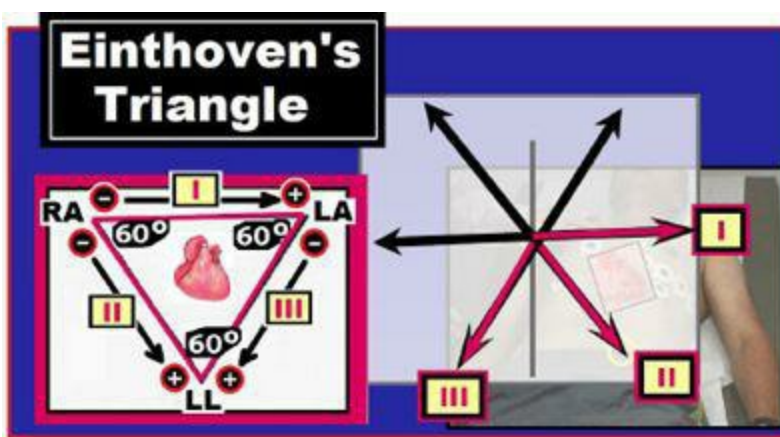


Figure 03.2-1: Electrically — the heart lies near the “center” of an *equilateral* triangle (*Einthoven’s Triangle*). Because *each* of the angles in an *equilateral* triangle has **60 degrees** — each of the **standard limb leads** (*leads I,II,III*) are separated from each other by 60 degrees. The ECG waveform for each of these limb leads is derived from the *difference* in electrical potential *between* 2 of the limb lead electrodes (*See text*).

Beyond-the-Core: In 2007 — an esteemed group of electrocardiography experts put forth an ACC/AHA/HRS *Scientific Statement of Recommendations for Standardization and Interpretation of the ECG* (Kligfield *et al.* — *JACC* 49:1109-1127, 2007). The stated goal of this consensus report was to reassess the relation of the resting ECG to its technology. Among the many insightful conclusions arising out of this publication (*many of which are described in this Section 3*) — is that the term, “unipolar” should *no longer* be used.

- In the past — it was said that 3 of the 12 leads on a *standard* ECG were “bipolar” (= *leads I,II,III*) — and the *other* 9 leads (*aVR,aVL,aVF*; and *V1-thru-V6*) were “unipolar”. *No longer!* The ACC/AHA/HRS Consensus Report emphasized that ***all* leads are effectively “bipolar”**.
- In the past — derivation of the 3 *augmented* leads (*aVR,aVL,aVF*) was determined from the difference in electrical potential between the respective anatomic recording site *minus* a central null vector. This process is no longer used. Instead — all 12 leads now use a *derived* electrode to serve as the *opposing* electrode for any lead pair.
- ***Chest leads*** use Wilson’s central terminal as the opposing *derived* electrode (*Section 03.6*). In contrast — the 3 ***augmented leads*** (*aVR,aVL,aVF*) use the Goldberger central terminal. The central terminal *previously* used during the early days of electrocardiography was derived from the mean potential of RA, LA and LL electrodes. As a result — the amplitude of complexes recorded from these extremity leads was low because the potential from the recording site was part of the central terminal. Thus, the recording potential was partially *subtracted* from the total potential displayed on the ECG. By *removing* the potential of the lead being recorded from the central terminal — Goldberger was able to ***augment*** the **amplitude** of recording complexes ***by 50%***.

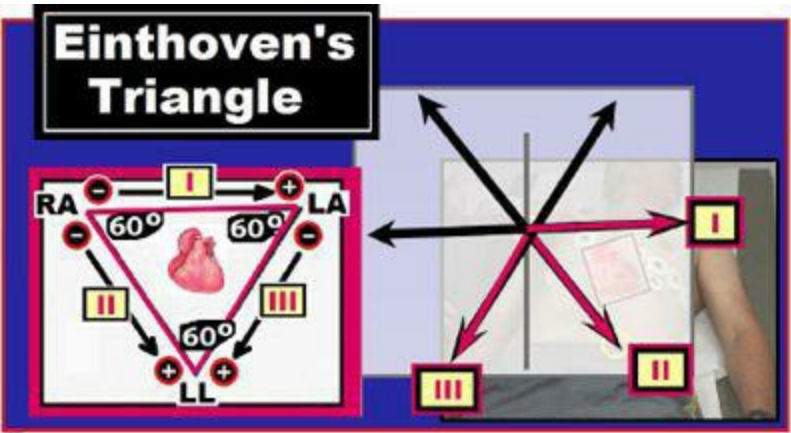
Abbreviations of Societies contributing to the Consensus Report:

- ACC — American College of Cardiology.
- AHA — American Heart Association.
- HRS — Heart Rhythm Society.

03.3 – Derivation of the *Standard Limb Leads (Leads I,II,III)*

As stated — the ECG waveform in the **3 standard limb leads** is derived from the *electrical difference* between 2 of the standard limb lead electrodes. From **Figure 03.2-1** — it can therefore be seen that the electrical potential of standard **lead I** is a result of the *electrical difference* between the LA and RA recording electrodes. Since the electrical wavefront travels from negative to positive — **positive Lead I** is directed *toward* the left arm in a horizontal plane that corresponds to an angle of **zero degrees**. As we will see momentarily (*in Section 03.5*) — zero degrees is our *starting point* in the *Hexaxial Lead System*

- It follows (*from Fig. 03.2-1*) — that the electrical potential of standard **Lead II** is the *difference* between the LL and RA electrodes.
- **Lead III** — is derived from the *difference* between the LL and LA electrodes.



Reproduced Figure 03.2-1: Einthoven's triangle. The ECG waveform for each of the 3 *standard* limb leads (*I,II,III*) is derived from the *difference* in electrical potential *between* 2 of the limb lead electrodes (*See text*).

Beyond-the-Core: The 4 *limb lead electrodes* (*placed on the RA,LA,LL and RL extremities*) — serve to define the 6 frontal plane limb leads.

- The **RL (*Right Leg*) electrode** — serves as an electronic reference that helps attenuate unwanted noise (*interference*). This **RL electrode** (*which is not shown in Fig. 03.2-1*) — is *not* used in the derivation of any of the limb leads.
- This leaves **3 pairs of electrodes** to generate the ECG waveforms of the 6 limb leads. Within *each* pair — one electrode is established as the *positive* end of the lead — whereas the *other* electrode of the pair is deemed the *negative* end. For example (*as seen in Figure 03.2-1*) — **Lead I** is derived from the electrical difference between the LA *minus* the RA electrodes (LA-RA), whereby the LA electrode is *positive* and the RA electrode is *negative*.
- Since the sum of voltage gains and voltage drops within the *closed* circuit of the 3 standard limb leads is equal to zero (*according to Einthoven's Law*) — the potential of **lead II** = the potential of **lead I** + **lead III** at *any* instant of time in the cardiac cycle.

- **KEY Point:** Because the potential of Lead II = Lead I + Lead III — any of the 3 *standard* limb leads can be mathematically derived from the *other* 2 leads. Therefore — the 3 standard limb leads contain *only* 2 independent pieces of information (*Kligfield et al. — JACC 49:1109-1127, 2007*).

03.4 – The 3 Augmented Leads (Leads aVR,aVL,aVF)

In addition to the 3 *standard* limb leads (I,II,III) — there are 3 **augmented limb leads**. These are **Lead aVR** — **Lead aVL** — and **Lead aVF**. These 3 extremity leads record the difference in electrical potential between the respective extremity lead site and the Goldberger reference central terminal (*Section 03.2*). For practical purposes — the orientation for each of these 3 **augmented leads** (aVR,aVL,aVF) can be thought of as an *outwardly* directed line extending from the electrical center of the heart to the particular electrode site on the right arm; left arm; or left leg (*outwardly directed red arrows in Figure 03.4-1*).

- **NOTE:** As discussed in Section 03.2 — Leads aVR, aVL and aVF are known as the "**augmented**" leads (*which accounts for the little "a" preceding VR, VL and VF*). The reason for this "a" — is that these extremity leads normally produce a relatively *small* electrical potential. This smaller electrical potential is magnified (ie, *augmented*) by approximately 50% through use of the Goldberger central reference system. Doing so results in comparable *relative* size for the QRS complex in all 12 leads of a standard ECG.

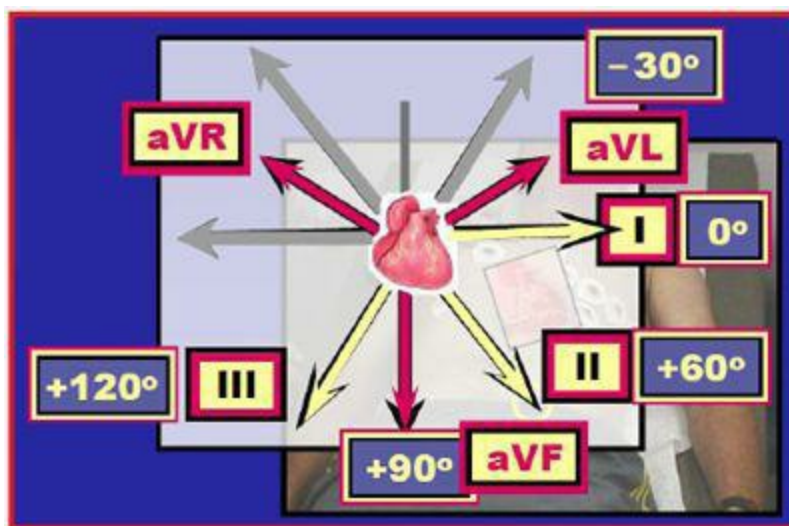


Figure 03.4-1: Hexaxial lead system with lead orientation in number of degrees displayed for the 6 limb leads. Electrical orientation for the 3 **standard limb leads** (I,II,III) — is depicted by *outwardly directly yellow* arrows at 0, +60 and +120 degrees. Orientation for the 3 **augmented leads** (aVR,aVL,aVF) — is depicted by *outwardly directly red* arrows. **Lead aVR** — is in the upper right quadrant; **lead aVF** — is vertical at +90 degrees; **lead aVL** — is in the upper left quadrant at -30 degrees (*See text*).

Beyond-the-Core: As alluded to in Section 03.2 — the Goldberger central terminal is now used as the opposing electrode for each of the extremity leads instead of Wilson's central terminal that was

used in the past. The Goldberger central terminal is derived by dividing the sum of the electrical potential of the other 2 extremity leads by two. That is, for **lead aVR** — this value is $(LA + LL)/2$; for **lead aVL** — it is $(RA + LL)/2$; and for **lead aVF** — it is $(RA + LA)/2$.

- **KEY Point:** It is *not* important to try to memorize these *hard-to-remember* relationships! **What IS important** — is to appreciate that the ECG waveform for *each* of the 3 *augmented* leads can be *derived* simply from knowing the ECG appearance of any 2 of the 3 standard limb leads! Thus, from a mathematical standpoint — there is ***much redundancy*** of information. Among the 12 standard leads that make up an ECG — there are *only* 8 independent pieces of information (*because 1 of the 3 standard limb leads and all 3 of the augmented leads are derived from the other 2 limb leads*)!
- **BOTTOM Line:** The AHA/ACC/HRS Consensus Report wants clinicians to recognize the *derived and redundant* nature of the 6 limb leads. Technically — only 2 leads are needed to reproduce all ECG waveforms in these 6 leads (*Kligfield et al. — AHA/ACC Recs. JACC 49:1109-1127, 2007*). That said — all 6 limb leads are retained because their use facilitates understanding and clinical interpretation (ie, *it would seem far more difficult and contrived to contemplate diagnosis of acute inferior MI from only seeing lead I and lead II — although this theoretically is possible*).

03.5 – The *Hexaxial* Lead System

Figure 03.4-1 puts together lead orientation for the 6 *limb* leads. This composite makes up what is known as the “*Hexaxial Lead System*”.

- **Lead I** — is a *lateral (leftward)* monitoring lead that views the heart's electrical activity from a *horizontally* oriented vantage point defined as **0 degrees**. This is our **starting point** in the *Hexaxial* Lead System.
- In contrast to lead I — **standard Lead II** and **Lead III** are **inferior leads** that respectively view the heart's electrical activity from vantage points angulated at **+60** and **+120 degrees** relative to the heart's electrical center.
- **Lead aVL** — is another *lateral (leftward)* monitoring lead (Figure 03.4-1). It records the heart's electrical activity from a vantage point that looks down at the heart from the patient's *left* shoulder (*from a position that corresponds to an angle of -30 degrees with respect to the heart's electrical center*).
- **Lead aVF** — is an *inferior* monitoring lead. The recording electrode is placed on the patient's *left* lower extremity — and the heart's electrical activity is viewed from a vantage point that looks directly up at the heart from the patient's *feet*. This provides a *perpendicular* vantage point (*that corresponds to an angle of +90 degrees*).
- **Lead aVR** — is the last of the augmented leads. It is the most distant recording electrode. Lead aVR views the heart from afar — looking down from the patient's *right* shoulder. Because the heart sits in the left side of the chest — the heart's electrical activity will generally be directed *away* from this *right-sided* and most distant recording electrode. As a result, for practical purposes — QRST morphology in lead aVR usually contributes *little* to interpretation of most

tracings (*with notable exception when there is lead misplacement or dextrocardia*). **PEARL:** There are some additional important clinical situations in which lead aVR may provide *invaluable* information to assist our interpretation (*We cover this concept in detail in Sections 09.31 thru-through 09.40 devoted to How to Use Lead aVR*).

03.6 – Precordial Lead Placement

In addition to 3 limb and 3 *augmented* leads — **6 more leads** are routinely included in a **standard 12-lead ECG**. These are the **precordial chest leads** — which view the heart's electrical activity in the **transverse (horizontal) plane**. These 6 precordial leads are positioned according to **anatomic landmarks** (Figure 03.6-1). As opposed to the 3 *augmented* limb leads (*aVR, aVL, aVF*) which are connected within a *closed* electrical loop — the 6 precordial *chest* leads are *independent* of each other (ie, *they are based on electrical activity assessed from their specific recording site* — and cannot be derived from other electrodes as the 6 limb leads can).

- The electrical recording from each chest lead is derived from the *difference* in potential between the respective anatomic recording site and a reference central terminal. For the chest leads — the central terminal used is the one first derived by Wilson in the 1930s. This central terminal is equal to mean potential from the RA, LA and LL electrodes (*roughly corresponding to the center of Einthoven's equilateral triangle*).
- **BOTTOM Line:** Each of the 6 precordial (*chest*) leads provides *independent* information based on electrical activity recorded from its respective anatomic site (*as shown in Figure 03.6-1*) — minus the potential from a central reference terminal. Clinically — the viewpoint of each *precordial* lead on a 12-lead tracing correlates with the anatomic area of the heart that lies below it.

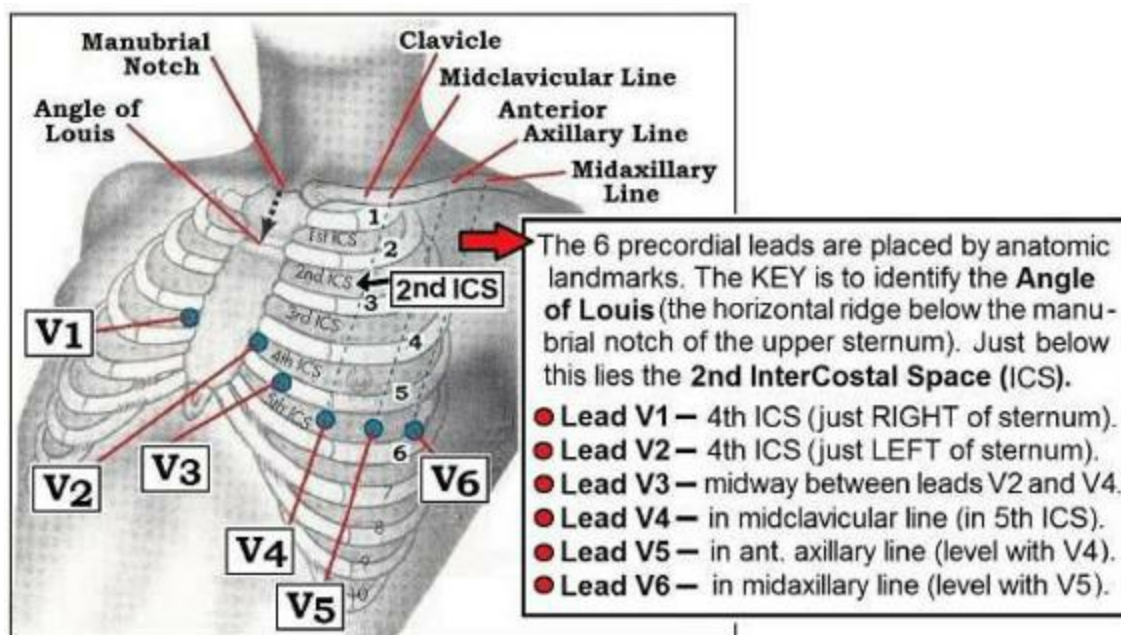


Figure 03.6-1: Anatomic landmarks for *precordial* lead placement (*See text*).

03.7 – Use of *Additional Leads*

Most of the time — the 12 viewpoints provided by a **standard 12-lead ECG** convey adequate information to *appropriately* evaluate and manage the patient. That said — there are occasions when use of **additional leads** may be helpful. Most commonly, these *extra* viewpoints include one or more *right-sided* leads (*especially lead V4R*) — and/or use of *posterior* leads (*V7, V8, V9, V10*).

- The goal of considering **additional leads** is to facilitate recognition of acute RV (*Right Ventricular*) and/or *posterior* infarction. This objective has resulted in preference by some for a **15-lead ECG** (*addition of V4R, V8, V9 to the standard 12 leads*).
- **Lead V4R:** — same placement as lead V4, but on the *right* chest.
- **Lead V8:** — placed in the back at the tip of the left scapula at the *same* horizontal level of lead V6.
- **Lead V9:** — placed in the back in the left paraspinal area in between the scapula and posterior spine, level with V6.

Our Bias: We prefer to **start** with **12 leads**. *Most* of the time — 12 leads are all that are needed to determine optimal *initial* management. That said — it is good to be aware of those situations for which use of *additional* leads may be helpful (*We return to this concept in Section 10.31 on acute RV MI — and in Section 10.36 on diagnosis of posterior MI*).

- **BOTTOM Line:** — Opinions vary regarding need for 12 vs 15 leads. Clearly, a balance must be struck between *urgency* of the situation — *How Long* it will take to obtain additional leads — and whether this time is really worth what you'll *actually* learn from getting more leads.

03.8 – *Technical Errors: Angle of Louis and Lead V1*

Accurate placement of **precordial leads** on the chest is essential. Placing a *precordial* lead as little as one IC (*Intercostal Space*) either too high or too low can dramatically alter QRS morphology and amplitude.

- The **KEY** to determining **correct placement** of precordial leads is to first identify the ***Angle of Louis***. This can be done by lowering your finger from the ***manubrial notch*** (*in the midline at the upper edge border of the sternum*) — until it comes to lie on a small ***horizontal ridge***. The **2nd intercostal space** lies just below this point (**Figure 03.8-1**).
- Drop down 2 additional intercostal spaces (*to the 4th intercostal space*) — and move *just to the right* of the sternum to locate the reference position for **Lead V1** (**Figure 03.6-1 and Figure 03.8-1**).

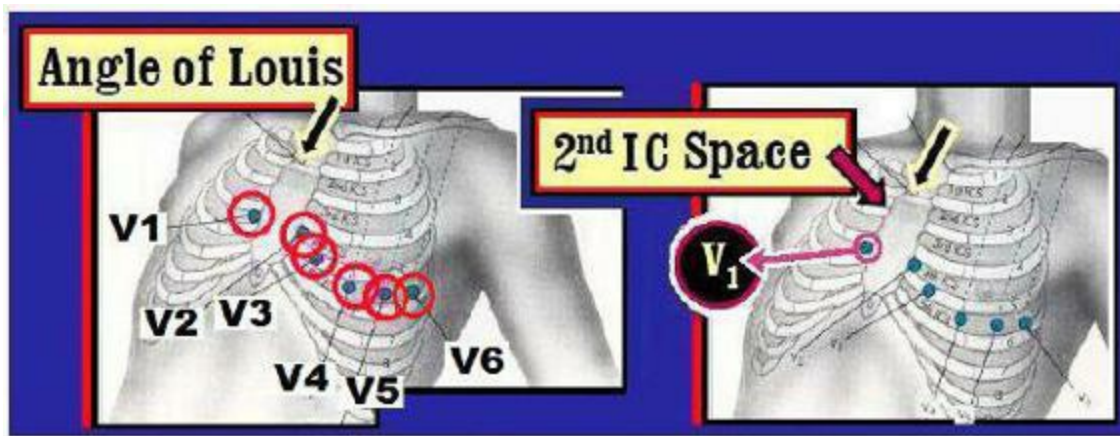


Figure 03.8-1: Anatomic landmarks (*Angle of Louis*).

- **Clinical NOTE:** Amazingly — *inaccurate* placement of lead V1 remains one of the most common technical errors committed. From description of *precordial* lead placement in [Figure 03.6-1](#) — it should be obvious that IF the *wrong* intercostal space is used for placement of lead V1 — that all precordial leads will be inaccurately placed!

03.9 – *Technical Mishaps: Important Caveats*

Be aware of the following caveats:

- In *women* — recording electrodes should be placed under the left breast to avoid precordial lead placement errors. This is especially problematic in women with large breasts. That said — consensus is lacking as to optimal electrode placement in women because of inconsistent literature results on the effect lead placement has on ECG waveforms. The amount of voltage attenuation by breast tissue appears to be variable — and reproducibility of precordial lead placement is often precarious, depending on experience of the technician; breast size, shape and adiposity; and small changes in patient position. **BOTTOM Line:** The AHA/ACC/HRS Consensus Statement still recommends electrodes be placed *under* the breast at this time — but this recommendation may change pending additional study (*Kligfield et al. — AHA/ACC Recs. JACC 49:1109-1127, 2007*). Until that time — the *KEY* is *consistency* of lead placement in any given patient!
- **Lead V5 and lead V6** should both be placed level with lead V4. This is *preferred* to the 5th intercostal space, which was the *previously* recommended reference point (*Kligfield et al. — AHA/ACC Recs. JACC 49:1109-1127, 2007*). The reason for recommending placement of leads V5 and V6 at the same horizontal level as lead V4 — is that the course of each intercostal space is variable.
- Definition of **lead V5** as *midway between V4 and V6* is a *more reproducible* landmark than the anterior axillary line — especially in cases when body habitus precludes accurate identification of the anterior axillary line (*Kligfield et al. — AHA/ACC Recs. JACC 49:1109-1127, 2007*).
- **Chest wall abnormalities** — are a common source of placement errors. These include pectus excavatum or carinatum (*hollow or pigeon breast*) — severe emphysema — and large body habitus, among others. Making a small mark on the chest where the leads were placed will hopefully clarify lead placement and promote consistency with *serial* tracings.

- **Is the Bed Flat?** — Often overlooked is the effect that *inclination* of the **bed** may have on the 12-lead ECG recording. Patients with **acute dyspnea** (*as is common with acute heart failure, COPD, pulmonary embolism*) — may simply not be able to lie flat when first seen. Inclination of the bed by 20-30 degrees may *either* “produce” Q waves or make Q waves disappear (*especially in the limb leads*). Be sure to note the *angle* of the bed when the ECG is recorded if it is not flat. This will hopefully resolve the problem of *inferior* Q waves “coming and going” as the patient gets better or worse.
- **Comparing Serial Tracings** — Remember to take into account the effect that a *change* in axis or precordial lead placement may have on the QRS and on ST-T waves. IF the mean QRS axis has *changed* between one tracing and the next — then the reason for a new Q wave seen on a subsequent tracing may be the *change* in axis (*and not necessarily the result of infarction*). Similarly — a change in precordial lead placement may dramatically alter QRS amplitude and morphology. The task of determining whether differences are due to a true ECG change vs axis shift/placement may be challenging!
- **Arm Leads** — should ideally be placed between shoulders and the wrists, away from bony prominences. **Leg Leads** — should be placed between hips and ankles, away from bony areas. IF for whatever reason more proximal lead placement (*of limb leads on the trunk*) is used — this should be noted on the tracing. *Consistency is key*.

03.10 – Important Concepts: Lead Misplacement/Dextrocardia

Although we include technical mishaps discussed in this Section 03 at an *early* point in this ECG ePub — many of these concepts are advanced. You’ll want to return to this section often ...

QUESTIONS on KEY Concepts:

- What might be wrong *technically* in **Panel A** and **Panel B** of Figure 03.10-1?
- How could you verify the cause for the *unexpected* appearance in each case?

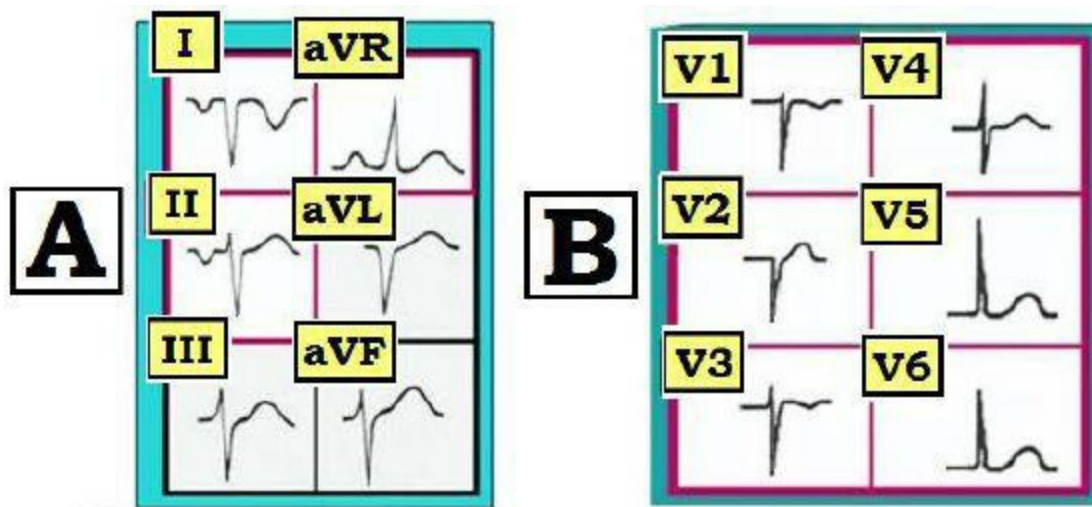


Figure 03.10-1: Which technical mishaps do you suspect? (*See text*).

Answer to Panel A in Figure 03.10-1: The most common error in lead placement is mixing up left

and right arm electrodes. Suspect this (*or dextrocardia*) — IF there is: i) **global negativity** in **lead I** (*negative P, QRS and T wave*); ii) an *upright* QRS complex in **lead aVR** — and iii) a *negative* P wave in **lead II**. All 3 of these findings are present in Panel A:

- Normally — the **QRS** in *left-sided Lead I* will be **upright** (*assuming the heart lies in the left side of the thorax*). Lateral infarction may produce a Q in lead I — but usually there is *at least some* R wave. **You should never see “global negativity” in lead I** (*unless there is dextrocardia or lead reversal*).
- **Lead aVR** — should normally show a **negative** QRS complex. The heart’s electrical activity normally moves toward the *left* — or away from *right-sided* lead aVR. About the only time you won’t see global negativity in lead aVR — is with *marked RVH* (*unless there is dextrocardia or lead reversal*).
- **Lead II** — should *always* show an **upright P wave** IF the rhythm is sinus. The path of electrical activity from the SA Node to the AV Node is virtually parallel to the +60 degree angle of lead II (*Section 02.3*). Therefore — IF the P wave in lead II is *not* upright — then the rhythm is *not* sinus (*unless there is dextrocardia or lead reversal*).

Panel B: We *reproduce* Panel B from Figure 03.10-1 below in Figure 03.10-2. Which technical mishap do you suspect? *Explain* your answer.

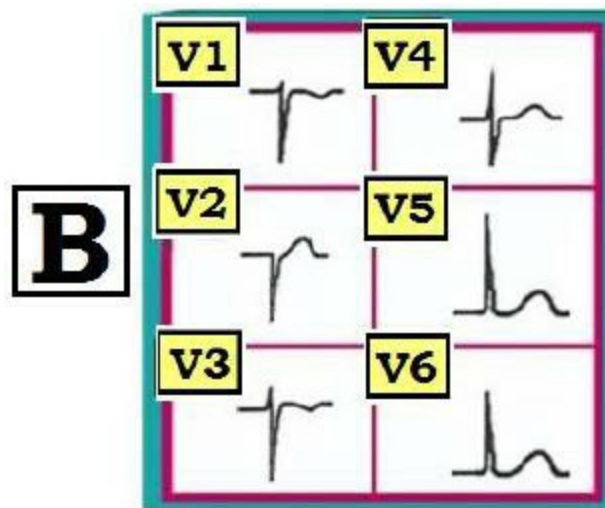


Figure 03.10-2: Which technical mishap do you suspect from the schematic *precordial* lead sequence shown here? (*See text*).

Answer to Figure 03.10-2: Lead V2 in the *precordial* lead sequence in Panel B simply does *not* make sense. There is not only *loss* of r wave from lead V1 to V2 — but an *upright* T wave is also seen between negative T waves in *neighboring* leads V1 and V3. *Suspect an error in precordial lead placement!*

- **Key Point:** *Unusual* R wave progression (ie, *abrupt transition*; *sudden loss or gain of R wave that only lasts for a single lead*) — should suggest precordial lead misplacement. IF ever in doubt — **Repeat the ECG!**

03.11 – Dextrocardia: *ECG Recognition*

Dextrocardia is rare — but it *does* occur. The average clinician will see *no more* than a handful of cases in his/her lifetime. Dextrocardia typically produces the **same ECG picture** in the limb leads as *right-to-left* arm lead reversal. That is — **lead I looks like aVR should look** (there is global negativity) — and lead aVR looks like lead I should look with a positive QRS complex (**Panel A** in Figure 03.11-1).

QUESTION on Recognizing Dextrocardia:

- IF the limb leads in **Panel A** of Figure 03.11-1 were the result of **dextrocardia** — Would you expect the *precordial* lead sequence to look more like **Panel C** or **Panel D**?
- Which *precordial* lead sequence would you expect IF the limb leads were the result of **limb lead reversal**?

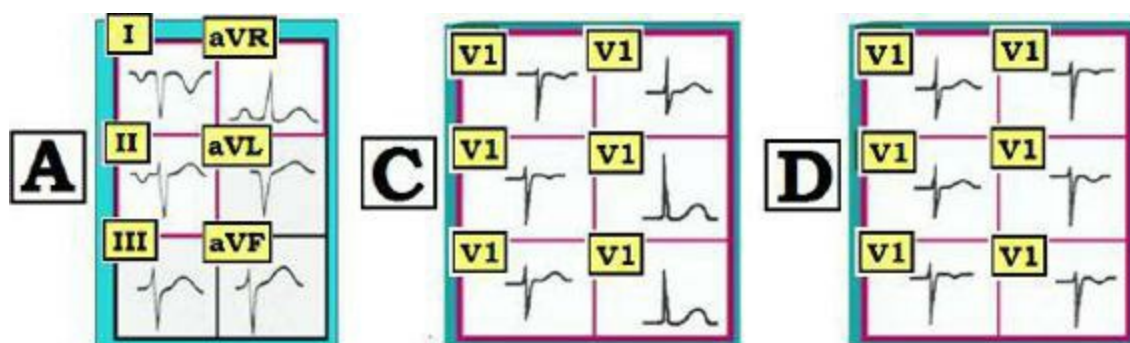


Figure 03.11-1: Limb lead appearance in **Panel A** is consistent with *either* limb lead reversal or dextrocardia. Which *precordial* lead sequence (**C** or **D**) would you expect if the patient had dextrocardia?

Answer to Figure 03.11-1: Panel C in Fig. 03.11-1 shows *normal* R wave progression in the *precordial* leads. Note gradual (*progressive*) increase in R wave amplitude as one moves from lead V1-toward-V4 with — transition (where the R wave becomes taller than the S wave is deep) occurring at normal location between V3-to-V4 (Section 09.5).

- IF the *limb* leads shown in **Panel A** were accompanied by the *precordial* lead sequence in **Panel C** — this would suggest simple **lead reversal** (because R wave progression is normal as expected when the heart lies in the left hemithorax).
- On the other hand — We would suspect **Dextrocardia** IF the *limb* leads in **Panel A** were accompanied by the *precordial* lead sequence in **Panel D** — because **reverse R wave progression** is seen. That is, the R wave is *tallest* in lead V1 of **Panel D** — and becomes progressively *smaller* as one moves across the *left* precordium. This is as one would expect IF the heart was in the *right* hemithorax.
- **Confirmation of Dextrocardia** is easy: **i)** Listen to the heart (*heart sounds will be heard on the right*); **ii)** Chest X-Ray will show a *right-sided* heart shadow; and/or **iii)** Repeat the ECG with precordial leads placed on the right — and you will now see *normal* R wave progression.
- **Beyond-the-Core:** There are many potential variations of dextrocardia, each with its own particular characteristics. Thus, the heart may lie on the right — but the atria and ventricles may

be arranged normally (*dextroversion*). Alternatively — the heart may be a *mirror* image of normal, with the atria and ventricles reversed. The great vessels and other visceral organs may or may not be reversed. The **P wave** in **lead II** may or may not be negative —depending on the nature of associated anatomic abnormalities. Regardless of P wave appearance in lead II — a form of dextrocardia should be suspected IF ever you see **global negativity in lead I with QRS positivity in lead aVR**. Confirmation can then be forthcoming by: **i)** listening on the right for heart sounds; **ii)** Chest x-ray; *and/or* **iii)** Repeating the ECG with precordial leads placed on the right (*which will now show normal R wave progression*).



PRACTICE Tracings:

Consider the following series of tracings (*Sections 03.13-thru-03.23*). In each case — Identify the likely technical mishap. *Our answers follow after each tracing.*

- **Be Aware:** — An example of dextrocardia is included.

Acknowledgements: My appreciation to the following people for allowing me to use their tracings that appear in the following Figures:

- David Richley (*of Scarborough in North Yorkshire, UK*) — Figures 03.16-1; 03.16-2; 03.17-1; 03.18-1; 03.21-1.
- Dawn Altman (*of the ECG Guru*) — Figures 03.19-1; 03.19-2.
- Jenda Enis Stros (*of Liberec, CzechRepublic*) — Figures 03.20-1; 03.22-1.

03.13 – PRACTICE: *Tracing A*

The rhythm in Tracing A was diagnosed as atrial flutter. *Do you agree?*

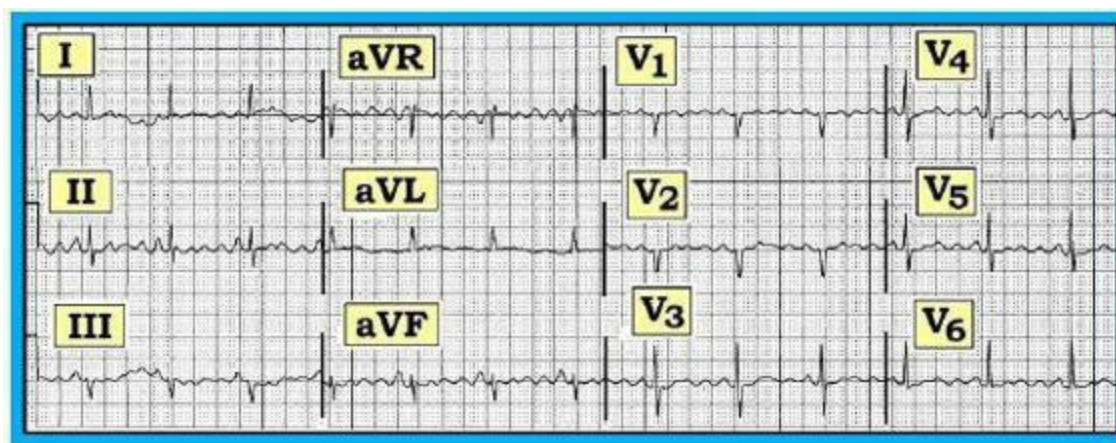


Figure 03.13-1: Practice Tracing A.

Answer to Tracing A: At first glance — it *looks like* there are flutter waves in lead II, as well as in several other leads on this tracing. That said — the rhythm is ***not* AFlutter** because: **i)** The undulations we see in lead II and elsewhere are not nearly regular enough to represent the 300/minute consistent “sawtooth” pattern of atrial flutter; **and ii)** We can see regular underlying P waves (*red arrows in Figure 03.13-2*).

- Note that the P waves highlighted by *red arrows* in Figure 03.13-2 are *unaffected* by the baseline artifact!
- Clinically — one look at the patient *confirmed* that the baseline activity was artifact from a marked *resting* tremor. **PEARL:** Be aware that the rate of a **Parkinsonian *resting* tremor** is often close to the rate of AFlutter.
- NOTE: We have already seen the lead II rhythm strip from this patient (*Section 02.25*).

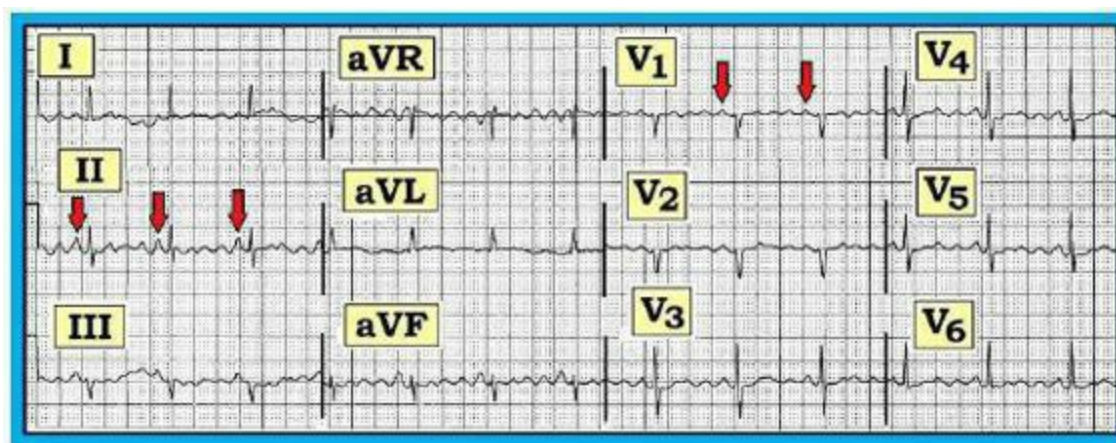


Figure 03.13-2: *Arrows* have been added to Figure 03.13-1 to highlight *underlying* sinus P waves. Baseline undulations are artifact from tremor.

03.14 – PRACTICE: *Tracing B*

Beats #6 and #7 were diagnosed as a ventricular couplet (2 PVCs in a row) — followed by 2 more PVCs (beats #10,12) later on in the tracing. *Do you agree?*

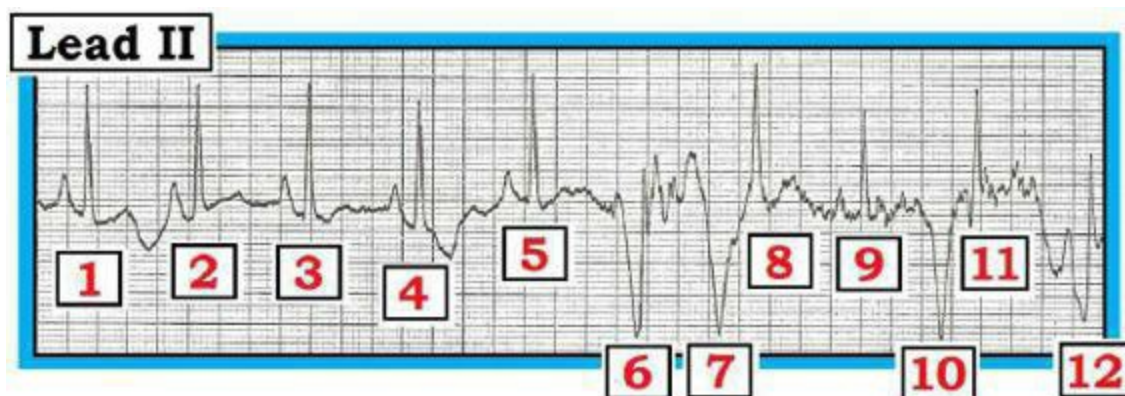


Figure 03.14-1: Practice Tracing B.

Answer to Tracing B: At first glance — it looks like there are *multiple* PVCs on this tracing. This is especially true for beat #6 — which is wide and perfectly placed where one might expect a PVC to occur. That said — we know that there are no PVCs on this tracing. Instead there is **artifact**. We say this because:

- There are baseline undulations seen throughout the tracing — especially during the last half of the tracing. Recognition that these coarse short vertical irregularities represent artifact should heighten awareness that other unexpected undulations might also represent artifact.
- The **timing** of **beat #7** is **impossible** for this beat to be a real. IF beat #7 was a PVC — there is *no way* that beat #8 could conduct (*since it would doubtlessly fall within the absolute refractory period of beat #7*). Knowing beat #7 is the result of **artifact** tells us that other *similar-looking* deflections (ie, #6,10,12) must *also* be artifact.

03.15 – PRACTICE: *Tracing C*

The rhythm in Tracing C was diagnosed as atrial flutter. *Do you agree?*

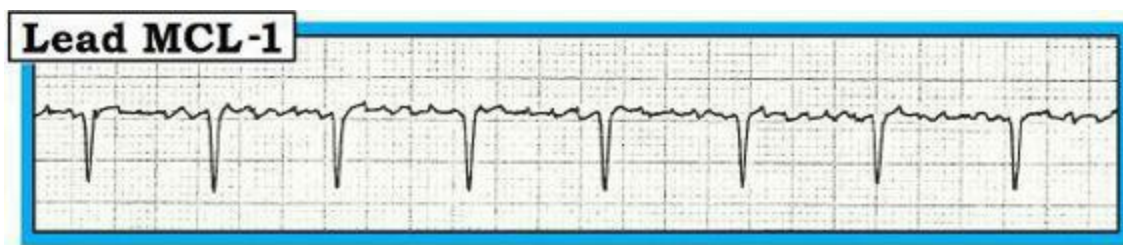


Figure 03.15-1: Practice Tracing C. (NOTE: **Lead MCL-1** is a right-sided monitoring lead that provides similar perspective as lead V1 on a 12-lead tracing).

Answer to Tracing C: As was the case for Tracing A (Figure 03.13-1) — the rhythm in Tracing C *initially* looks like AFlutter. That said — this rhythm is *unlikely* to be AFlutter because:

- A true sawtooth pattern is absent. Instead we see *irregularly* occurring short vertical spikes at a rate significantly *greater* than 300/minute. This strongly suggests that these short vertical spikes represent **artifact**.
- NOTE: We are *not* at all certain what the rhythm in Figure 03.15-1 is. A **12-lead ECG** would be needed to comment further — as this will reveal IF flutter or other atrial activity is seen in other leads. What *can* be said from **Tracing C** in Fig. 03.15-1 is the following: **i)** The rhythm looks to be supraventricular (*narrow QRS in this MCL-1 lead*); **ii)** The rhythm is almost but *not* completely regular (*slight but definite variation in R-R intervals of some beats when measured with calipers*); **iii)** No definite P waves are seen (*although we can't be sure if atrial activity is or is not present elsewhere without a 12-lead tracing*); and **iv)** **Artifact** appears to be present. Pending results of a 12-lead ECG — We *suspect* the rhythm is **probably AFib** (*Atrial Fibrillation*) given *lack* of P waves and *slight-but-definite* irregularity in the rhythm.

03.16 – PRACTICE: *Tracing D*

What *might* be wrong?

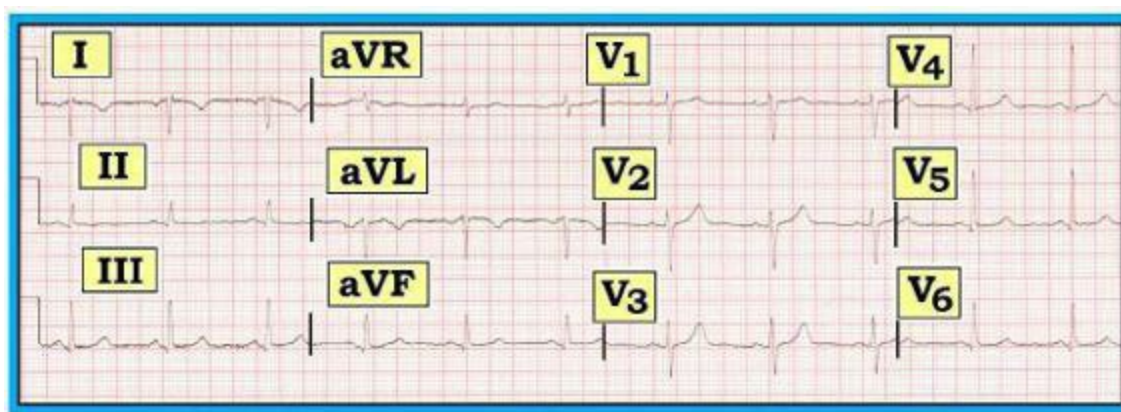


Figure 03.16-1: Practice Tracing D.

Answer to Tracing D: There is **global negativity** in lead **I** (of the P wave, QRS and T wave). This should virtually *never* occur normally. The most likely causes of global negativity in lead I are: **i)** LA-RA (*Left Arm-Right-Arm*) lead reversal; **and ii)** Dextrocardia (Sections 03.10 and 03.11).

- In addition to *global* negativity in lead I — Note that the P wave, QRS complex and T wave in lead **aVR** are *not* negative as they usually are under normal circumstances. This is consistent with *either* lead reversal or dextrocardia.
- **R wave progression** is *normal* in *Tracing D* (with transition occurring between lead V3-to-V4). This defines the problem as **limb lead reversal** and *not* dextrocardia (Section 03.11).
- The diagnosis of **LA-RA limb lead reversal** was *confirmed* by **repeating** the ECG *after* verifying correct limb lead placement (**Figure 03.16-2**). Note in *Fig. 03.16-2* that the P wave, QRS and T wave in lead I are now all upright — **and** that there is now global negativity in lead aVR as is normally expected.
- **Clinical Note:** The P wave in lead **II** will often (*but not always*) be negative when there is *either* lead reversal *or* dextrocardia. That said — the P wave in lead II of *Figure 03.16-1* is *upright* despite limb lead reversal. The P wave is even more upright with correct lead placement (*Figure 03.16-2*).

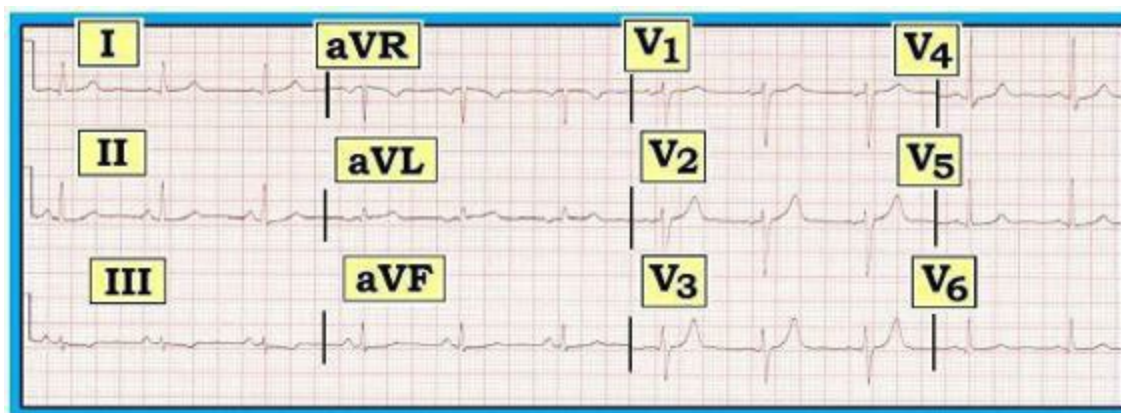


Figure 03.16-2: The ECG from *Figure 03.16-1* has been **repeated**. Note that the P, QRS and T wave in lead **I** are now positive — **and** that global *negativity* has been restored in lead **aVR**. R wave

progression is normal and unchanged from Fig. 03.16-1. This confirms that the unusual picture initially seen in **Figure 03.16-1** was due to **LA-RA limb lead reversal** (*and not dextrocardia*).

- Acknowledgement: My appreciation to David Richley (*of Scarborough in North Yorkshire, UK*) for allowing me to use these tracings.

03.16.1 – ADDENDUM: *Prevalence/Types of Limb Lead Errors*

There are *many* possible ways to misconnect the 4 limb lead electrodes. Of the *estimated* 300 million ECGs obtained annually worldwide — as many as 6 million ECGs (*~2% of the total*) are thought to manifest *some* technical mishap (*Rowlands DJ: J Electrocardiology 41:84-90, 2008*). Awareness of how to recognize technical mishaps when they occur is therefore essential!

- There are 6 ways to commit a *single* limb lead misconnection (*RA-LA; RA-RL; RA-LL; LA-RL; LA-LL; and RL-LL*). The number of possible errors increases if *multiple* lead mix-ups are included among the possibilities. Recall of the specific ECG pattern likely to be seen by each of these technical mishaps is challenging even for the advanced interpreter. Fortunately — it is *not* necessary to recall each particular ECG pattern *as long as* you recognize that one or more leads are misconnected.
- **NOTE-1:** The most common form of lead misconnection is the easiest to recognize. This is **LA-RA** (*Left Arm-Right Arm*) **limb lead reversal** — in which the ECG waveform in lead I looks like aVR, and vice versa (*Sections 03.10, 03.11 — and Figure 03.16-1*).
- **NOTE-2:** Lead misconnections as well as anatomic errors in placement may also occur in precordial leads. We address **precordial lead errors** later in this section (*Sections 03.20-thru-03.23*).

03.16.2 – ECG Findings that *Suggest Limb Lead Misconnection*

As stated — limb lead reversal is surprisingly common. Its occasional occurrence is an almost *inevitable* consequence of the quantity of ECGs that are ordered. Clinically — A **possible limb lead misconnection** should be *suspected* IF *any* of the following findings are present:

- **Global negativity** in lead I.
- A predominantly **positive QRS** complex in lead aVR.
- A **negative P wave** in lead II.
- A **null vector** (*completely flat line*) in *any* of the limb leads.

BOTTOM Line: It is prudent to suspect *possible* lead misplacement on recognition of any of the above findings. The “good news” — is that it is usually *easy* to check out IF your suspicion is accurate: Simply **repeat the ECG** to see if the abnormal finding goes away.

- Apply these concepts to the ECG examples we show in Sections 03.17-thru-03.19.

03.17 – PRACTICE: *Tracing E*

What *might* be wrong?

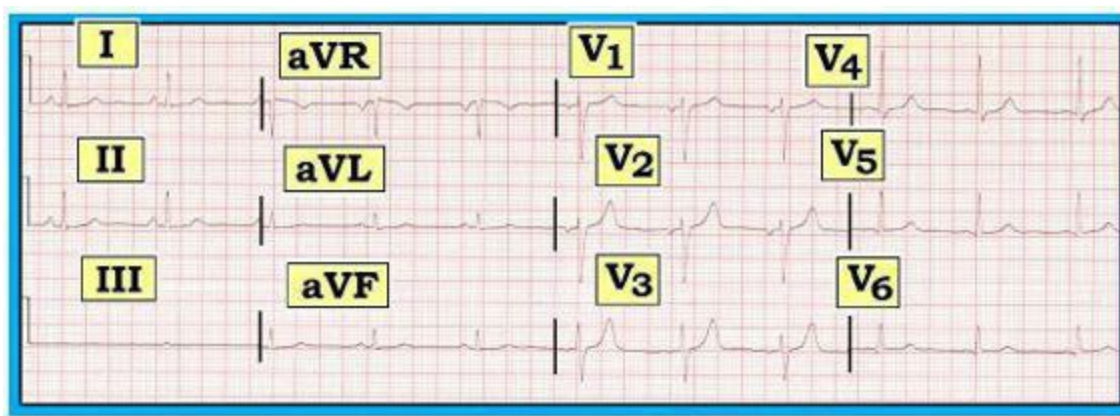


Figure 03.17-1: Practice Tracing E.

Answer to Tracing E: A **null vector** (totally flat line) is seen in **lead III**. This should virtually *never* occur normally. The most likely cause of a null vector in lead III, but an otherwise fairly normal tracing — is **LA-RL** (Left Arm-Right Leg) **limb lead reversal**.

- As emphasized in the beginning of this Section (in [Figure 03.1-1](#)) — the **RL** (Right Leg) **electrode** serves as a *zero* electrical potential (*ground lead*) reference point.
- Lead misconnections involving the RL electrode typically produce a *predictable* ECG pattern. With **LA-RL lead reversal** — Leads I and II look the same; leads aVL and aVF look the same — and **lead III** records a **null vector**. This is *precisely* the pattern we see in [Figure 03.17-1](#). Note that *precordial* lead R wave progression in [Fig. 03.17-1](#) is normal (with transition occurring between V3-to-V4).
- IF on the other hand, there was **RA-RL lead reversal** — Leads I and aVL will look the same (with a *negative QRS*); leads aVR and aVF look the same (with a *positive QRS*) — and it will be **lead II** that records a **null vector** ([Figure 03.18-1](#)).

BOTTOM Line: Many “brain cells” are required to commit the expected *specific* ECG patterns of LA-RL and RA-RL lead reversal to memory. This is *not* needed clinically — because ***all that counts is recognition that some type of lead misconnection is possible***. Awareness of the 4 *easy-to-remember* ECG findings cited in Section 03.16.2 should be all that is needed to recognize most clinically significant lead misconnections. Look for one or more of the following: **i)** *Global* negativity in lead I; **ii)** A predominantly *positive* QRS complex in lead aVR; **iii)** A *negative* P wave in lead II; and/or **iv)** A *null vector* (completely flat line) in any of the limb leads.

- ***Beyond-the-Core:*** The electrical potential recorded from the **RL** electrode is virtually *identical* to that recorded from the **LL** electrode (*zero in each case*). As a result — **RL-LL misconnection** does *not* change the relationships in Einthoven’s triangle — because *both* RL and LL electrodes record a similar *zero* electrical potential. This means you will *not* be able to recognize RL-LL misconnection based on ECG appearance. That said — this does *not* matter, because the 12-lead ECG will be essentially *unchanged* if RL and LL electrodes are inadvertently misconnected.

- Acknowledgement: My appreciation to David Richley (*of Scarborough in North Yorkshire, UK*) for allowing me to use this tracing.

03.18 – PRACTICE: Tracing F

What *might* be wrong?

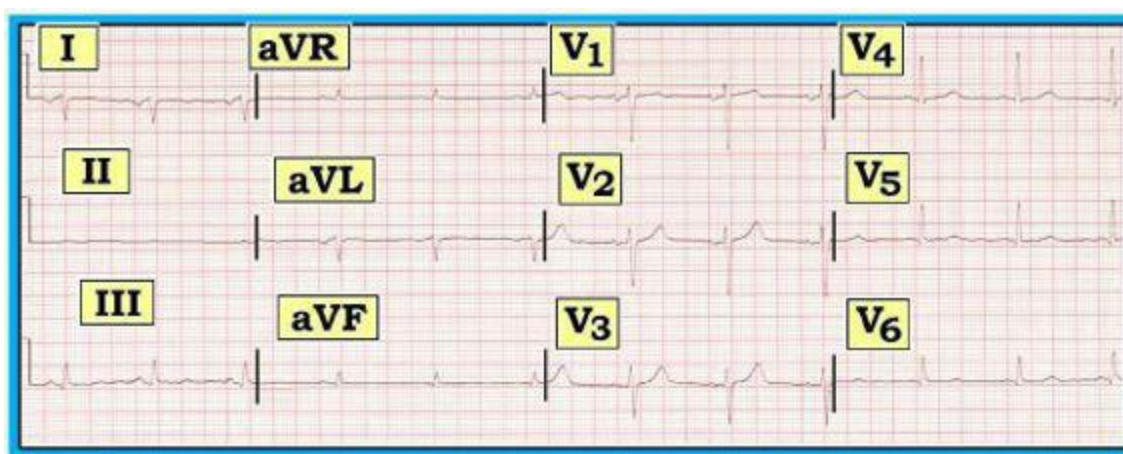


Figure 03.18-1: Practice Tracing F.

Answer to Tracing F: A **null vector** (totally flat line) is seen in **lead II**. This should virtually *never* occur normally. The most likely cause of a null vector in lead II — is **RA-RL** (*Right Arm-Right Leg*) **limb lead reversal**.

- In Section 03.17 — We discussed the ECG pattern expected with LA-**RL** lead reversal (Tracing E = Figure 03.17-1). In this case — the overall ECG looks relatively normal with exception of a null vector in lead III.
- In contrast — the **null vector** in **Figure 03.18-1** is seen in **lead II**. This suggests that the misconnection is the result of **RA-RL** lead reversal. Other features of RA-**RL** misconnection are that: **i)** Leads I and aVL look the same (*and manifest a negative QRS*); **and ii)** leads aVR and aVF look the same (*and manifest a positive QRS*). Note that precordial lead R wave progression in Fig. 03.18-1 is normal (*with transition occurring between V3-to-V4*).

BOTTOM Line: Once again — recall of the specific features that distinguish RA-**RL** lead reversal from other limb lead misconnections is far *less* important than recognizing **that some type of lead misconnection is likely**. Recognition of a technical mishap is *easy* for the ECG shown in Figure 03.18-1 because: **i)** there is global *negativity* in lead I; **ii)** lead aVR is positive; **and iii)** there is a *null vector* in one of the limb leads (*seen here in lead II*).

- **Confirmation** that limb lead reversal is the cause of the ECG picture in Figure 03.18-1 — is readily attained by *verifying lead placement* **and repeating the ECG**. The *abnormal* findings in Fig. 03.18-1 (*null vector in lead II; global negativity in lead I; positive R wave in aVR*) — should *no longer* be seen once an ECG with *correct* lead placement is obtained.
- **Acknowledgement:** My appreciation to David Richley (of Scarborough in North Yorkshire, UK) for allowing me to use this tracing.

03.19 – PRACTICE: *Tracing G*

What *might* be wrong?

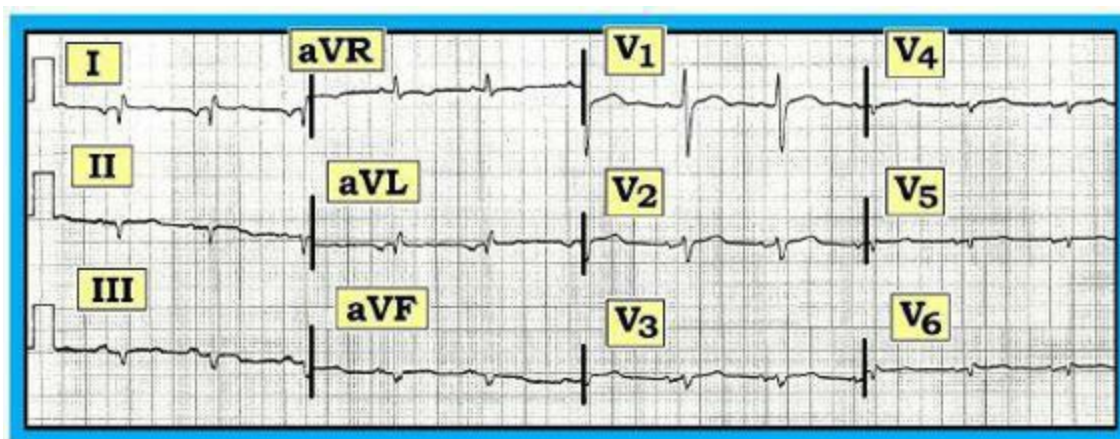


Figure 03.19-1: Practice Tracing G.

Answer to Tracing G: There are several distinctly *unusual findings* on the 12-lead ECG seen in Figure 03.19-1. These include:

- Very *negative P wave* with *deep Q wave* in **lead I**. This is *not* the obvious global negativity appearance that we have previously seen in [Figure 03.16-1](#) and [Figure 03.18-1](#) — in which we *knew* something was “wrong”. *But* — the P wave in *left-sided* lead I is usually *not* decidedly negative when the P wave is positive in lead II as it is here. In addition — the Q wave in lead I is clearly *too deep* to be a normal “septal” q wave. While possible the Q waves in leads I and aVL represent prior high lateral infarction — the *possibility* of a **technical mishap** should be considered.
- *Both* the P wave and QRS complex in **lead aVR** are positive. This *shouldn't* normally be if all leads are correctly placed *unless* there is marked underlying structural abnormality.

Impression of Figure 03.19-1: In addition to the *unusual* appearance of leads I and aVR — overall QRS amplitude in [Fig. 03.19-1](#) is greatly reduced (*with the largest QRS complex surprisingly being recorded in lead V1*) — there is marked frontal plane axis deviation — and precordial R wave progression is reversed. **Bottom Line:** Something is *very wrong* here.

- Our first reaction to seeing the ECG in [Figure 03.19-1](#) — is to find out more about the patient. *Are there symptoms?* Any history of heart disease or prior infarction?
- Is there a *prior* ECG?
- What does physical examination show? Are heart sounds heard on the *left* side of the chest?
- Is there a chest x-ray?
- What does a **repeat ECG** look like after verifying that all leads are correctly placed?

Follow-Up to the Case: Heart sounds were heard on the *right* side of the chest. The ECG was repeated with *precordial* leads placed on the *right* (**Panel B** in [Figure 03.19-2](#)). The patient had *dextrocardia*.

- As discussed in Sections 03.10 and 03.11 — there are *many* potential forms of dextrocardia. The common “theme” on ECG — is that the appearance of leads I and aVR is *reversed* with dextrocardia from what you would normally expect. This is indeed the case in [Figure 03.19-1](#).
- It is easy to **confirm dextrocardia** ([Section 03.11](#)): **i)** Heart sounds are heard on the right; **ii)** Chest x-ray shows a *right-sided* heart shadow; **and iii)** **Repeat ECG** with precordial leads placed on the right at least partially *normalizes* R wave progression. While admittedly, R wave progression in **Panel B** of [Figure 03.19-2](#) is not “normal” (*since a predominant R wave is never seen*) — *right-sided* placement of precordial leads clearly results in an *increase* in positive forces, as would be expected if the heart was situated on the *right* side of the chest.

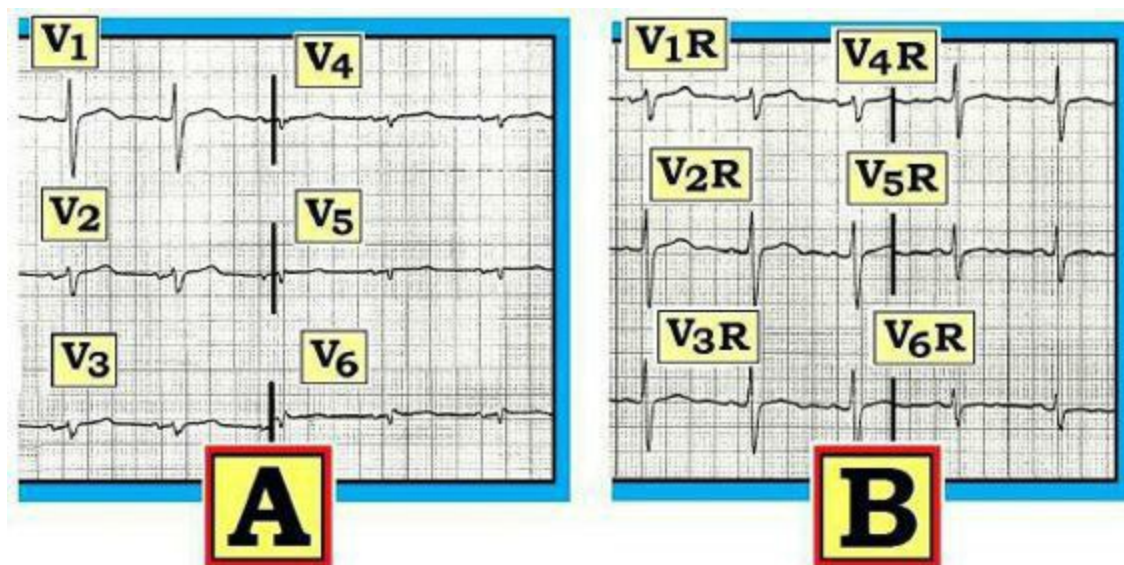


Figure 03.19-2: Comparison between the *original* precordial lead sequence seen in [Figure 03.19-1](#) (**Panel A**) — with the precordial lead sequence on *repeat* ECG with precordial leads now placed in comparable anatomic location, but on the *right* side of the chest (**Panel B**). Note that while a predominant R wave is never attained in **Panel B** — there is nevertheless a decided *increase* in positive QRS amplitude that supports the diagnosis of **dextrocardia**.

- Acknowledgement: My appreciation to Dawn Altman (*of the ECG Guru*) for allowing me to use these tracings.

03.20 – PRACTICE: *Tracing H*

Beat #5 was interpreted as either a PVC or an *aberrantly* conducted PAC. *Do you agree?*

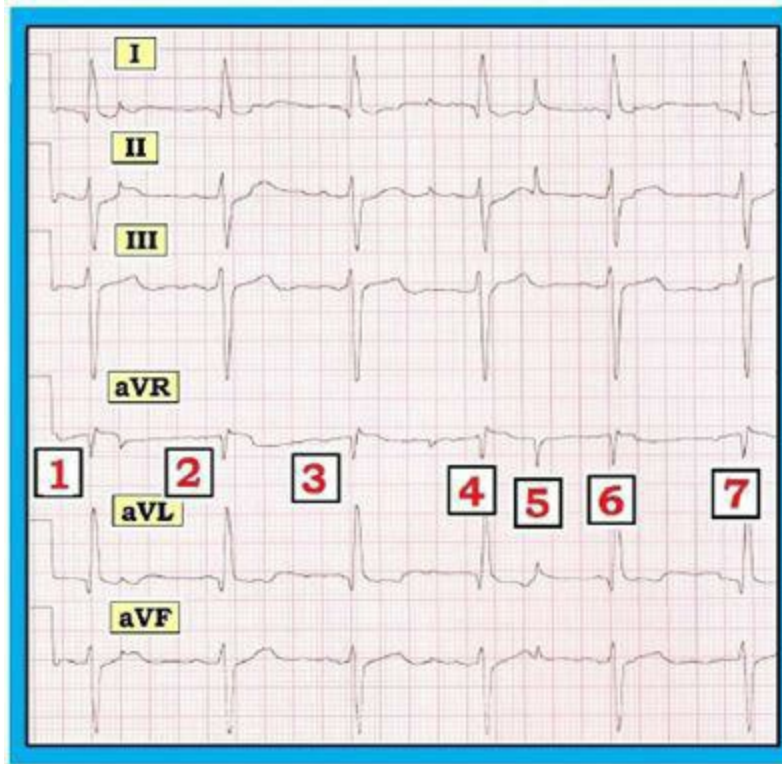


Figure 03.20-1: Practice Tracing H.

Answer to Tracing H: At first glance — **beat #5** looks like a legitimate early beat, especially in leads I, II and aVR. That said — beat #5 is not “real”. Instead, it is **artifact**. We know this because:

- Beat #5 *lacks* a T wave.
- There is absolutely *no effect* of the waveform labeled #5 on the underlying rhythm that continues *undisturbed* at the same R-R interval.
- The waveform labeled #5 *looks like* artifact.
- There is evidence *elsewhere* on this tracing of similar artifactual distortion. Note the small geometric spike in the T wave of beat #1 in leads II and aVR — as well as a similar small spike artifact occurring just past the midpoint of the R-R interval between beats #3-to-4 in leads I, II and aVR.

Bottom Line: The deflection labeled #5 in Figure 03.20-1 is not real. We can dismiss it as **artifact** *without* need for further attention.

- Acknowledgement: My appreciation to Jenda Enis Stros (*of Liberec, CzechRepublic*) for allowing me to use this tracing.

03.21 – PRACTICE: *Tracing I*

The *precordial* lead sequence seen in **Panel A** and **Panel B** of Figure 03.21-1 was obtained *moments apart* from the *same* patient.

- Can you guess what changed from one tracing to the next?

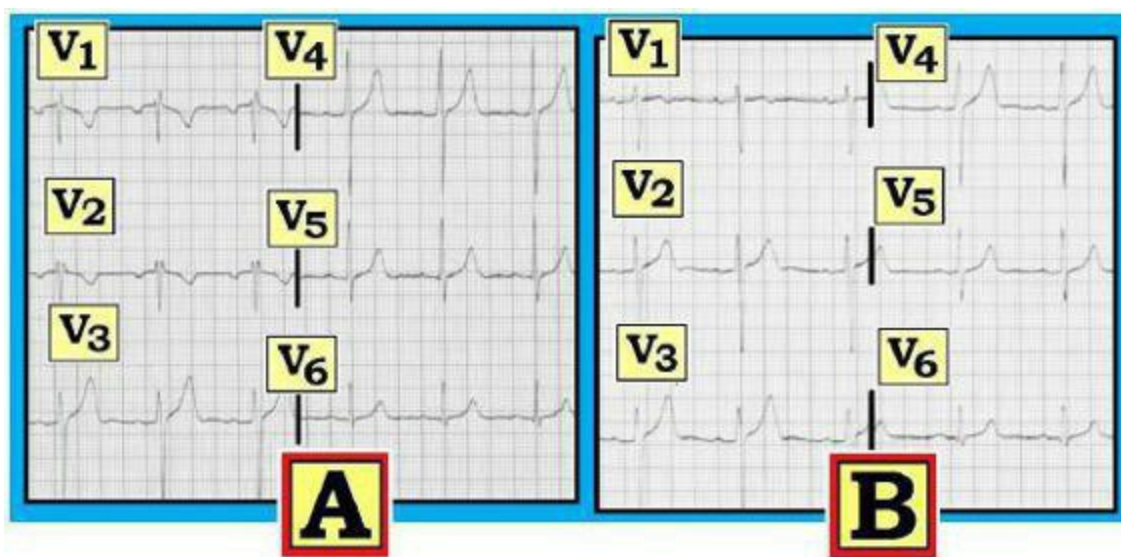


Figure 03.21-1: Practice Tracing I.

Answer to Tracing I: The *precordial* lead sequence in **Panel A** was obtained with **incorrect *high* placement** of several *chest lead electrodes*. The effect this may have on the ECG recording is obvious when compared to the **repeat** ECG performed on this patient a short while later with *verified* correct lead placement (**Panel B**).

- Note *loss* in amplitude of the *initial* r wave in leads V1 and V2 when chest leads are placed too high (**Panel A**). In addition — an rSr' (*incomplete RBBB*) pattern with T wave inversion was seen in *both* leads V1,V2 in Panel A — *but* is *no longer* seen in **Panel B** when the ECG is repeated with *correct* chest lead placement.

Clinical Note: The importance of correct chest lead placement *cannot* be overstated (*Sections 03.6 and 03.7*). Erroneous placement of lead V1 at a location that is 1 or 2 interspaces *too* high — will result in erroneous placement of the remaining 5 precordial leads. Potential consequences include:

- Misinterpretation of LAA (*Left Atrial Abnormality*) — since inadvertent high chest lead placement may result in *deepening* of the negative component of the P wave in lead V1.
- *Inaccurate* diagnosis of incomplete RBBB with associated ST-T wave abnormality (**Panel A** in Fig. 03.21-1). This could result in confusion with Brugada syndrome variants that are really *not* present (Section 05.XXXX)
- *Inaccurate* interpretation of R wave progression in the precordial leads — leading to overcall or undercall of prior anteroseptal infarction.
- *Inaccurate* representation of true precordial lead amplitude (*leading to inaccurate diagnosis of*

LVH).

- *False* impression that there has been a “change” in ECG appearance when either the prior tracing(s) or current tracing was *unknowingly* obtained with inaccurate chest lead placement.
- Acknowledgement: My appreciation to David Richley (*of Scarborough in North Yorkshire, UK*) for allowing me to use this tracing.

03.22 – PRACTICE: *Tracing J*

What *might* be wrong?

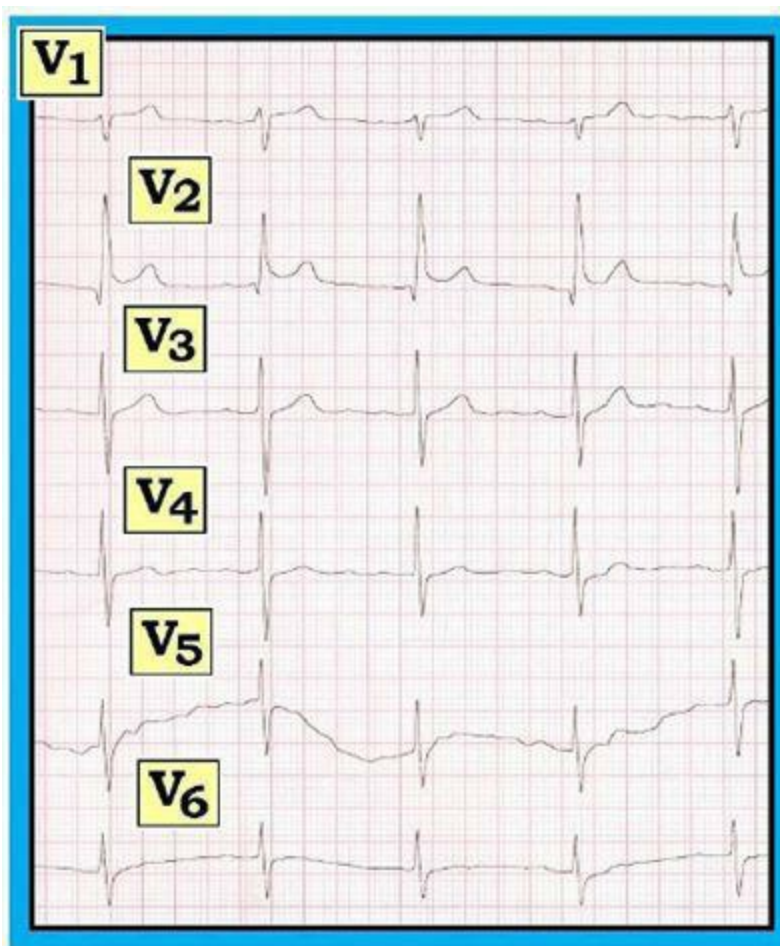


Figure 03.22-1: Practice Tracing J.

Answer to Tracing J: The precordial lead appearance of **lead V2** makes *no* sense:

- The initial small r wave that was seen in lead V1 has been lost. Instead — there is now a *small-but-definite* Q wave in **lead V2**. This is associated with a *disproportionately* tall R wave as well as some ST segment elevation.
- Equally *abrupt* change in QRST appearance between leads V2-to-V3 *as was seen* between leads V1-to-V2.

Bottom Line: *Something just looks wrong.* The solution is easy: **Repeat the ECG**, being sure to *verify* correct precordial lead placement. Only in this way can we determine IF the ST elevation seen in lead V2 of Figure 03.22-1 is likely to be real and of concern, or a consequence of inaccurate chest lead placement.

- Acknowledgement: My appreciation to Jenda Enis Stros (*of Liberec, CzechRepublic*) for allowing me to use this tracing.

03.23 – PRACTICE: *Tracing K*

What *might* be wrong?

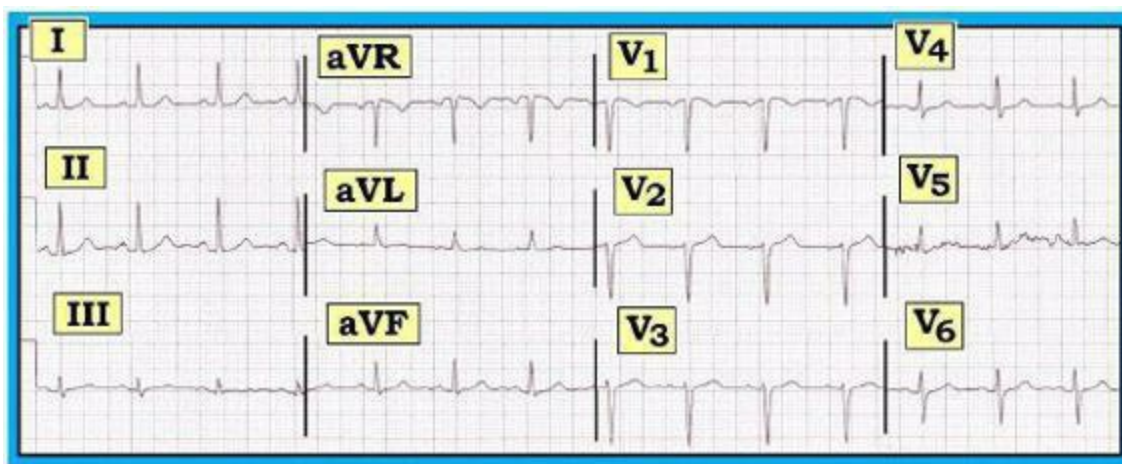


Figure 03.23-1: Practice Tracing K.

Answer to Tracing K: While overall the ECG in Figure 03.23 appears to be fairly unremarkable — **lead V6** simply does *not* make sense.

- We interpret the ECG in Fig. 03.23-1 as showing normal sinus rhythm; normal intervals and axis; no chamber enlargement; no more than a tiny q wave in lead III; normal R wave progression (*with transition between leads V3-to-V4*) — and some baseline artifact (*especially in lead V5*) but *no* acute ST-T wave changes. That said — it does not make sense for the S wave that disappeared after lead V4 to abruptly reappear with a predominant S>R wave in lead V6. Instead — it would make much more sense if the waveform currently seen in lead V6 was recorded in lead V4 (*with the current V4 and V5 recordings being instead from V5 and V6*).

Bottom Line: Simply *repeating the ECG* after verifying chest lead placement is an *easy* way to know for certain what the *true* precordial lead ECG appearance in **Tracing K** should be.

04.0 – Intervals (PR/QRS/QT)



Using the Systematic Approach: **–Rate – Rhythm... –Intervals–**

04.1 – What are the 3 Intervals in ECG Interpretation?

As per the **Systematic Approach** introduced in Section 01.1 — *after* assessing a 12-lead ECG for Rate *and* Rhythm — one then looks to determine the **3 ECG Intervals** (Figure 04.1-1):

- The **PR interval** — is defined as the period that extends from the *onset* of *atrial* depolarization (*beginning of the P wave*) — until the onset of *ventricular* depolarization (*beginning of the QRS complex*).
- The **QRS complex** itself (*discussed more in Section 05.1*).
- The **QT interval** — the period from the onset of the QRS complex until the end of the T wave (*See Section 06.1*).

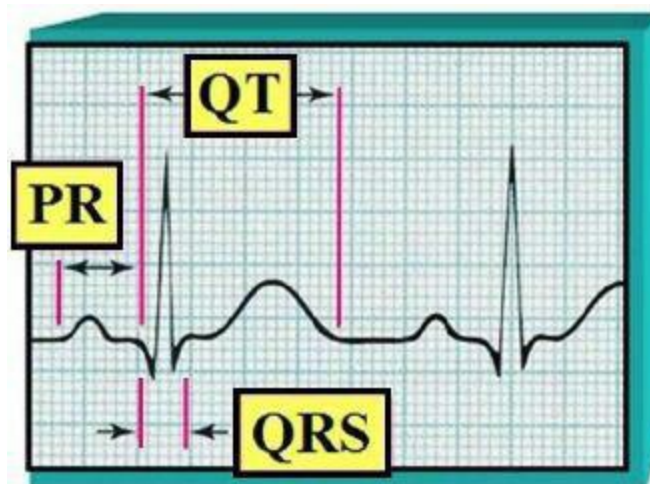


Figure 04.1-1: The 3 ECG Intervals (= the PR – QRS – QT intervals). Measure intervals in the lead where the interval looks *longest*. Precise determination of an interval is usually *not* necessary if it falls within the normal range.

04.2 – The PR Interval: *What is Normal?*

The *best* lead to assess the PR interval in is usually **lead II**. The P wave should be **upright** in lead II if the rhythm is sinus. In adults — the PR interval is considered **normal** if it measures *between 0.12 and 0.20 second* (*See Panel A in Figure 04.2-1*).

- The **PR interval** is "**short**" — if it measures *less* than **0.12 second** in duration in lead II (**Panel B**). One cause of a *short* PR interval is WPW (Wolff-Parkinson-White) syndrome in which an AP (Accessory Pathway) exists that *bypasses* the AV node, thereby requiring *less* time for the impulse to arrive in the ventricles (*See Section 05.37*). **NOTE:** *Not* all patients with a short PR interval (0.12 sec.) have WPW. Instead — the PR interval may sometimes be short because it is

anatomically small or conduction is fast. *Beyond-the-Core*: The chance that a patient has an AP becomes greater when the PR interval is very short (ie, *0.10 second*).

- The **PR interval** is “*long*” — if it measures *more* than **0.20-0.21 second**. One can tell at a glance if the PR interval is long — by looking to see IF it is clearly *more* than 1 *large box* in duration (**Panel C** in Fig. 04.2-1).

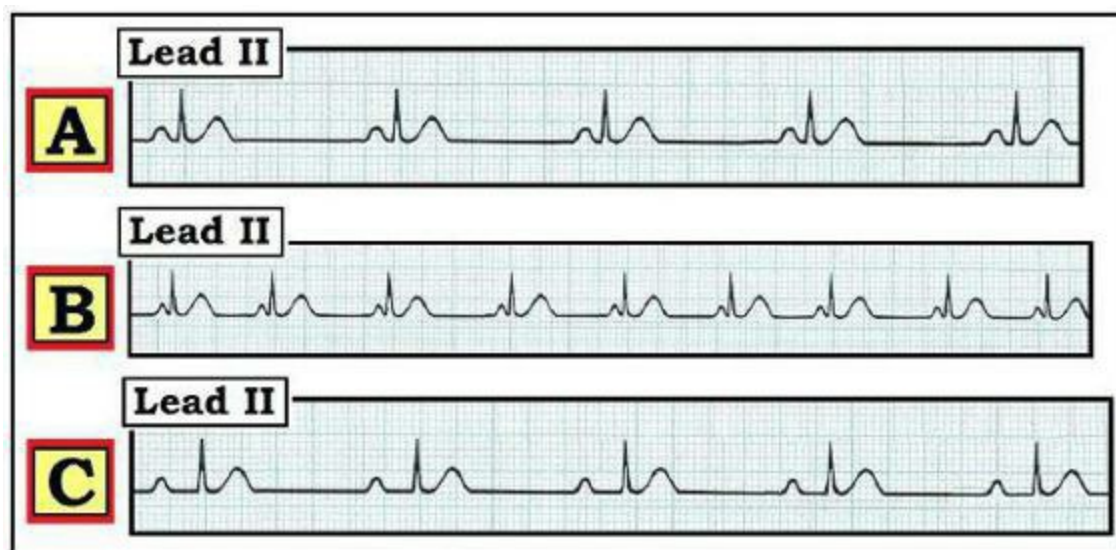


Figure 04.2-1: Limits of the PR interval. **Panel A** — The PR interval is normal (*between 0.12-0.20 second in adults*). **Panel B** — the PR interval is short (*less than 0.12 second*). **Panel C** — the PR interval is long (*clearly more than one large box in duration*).

04.3 – The PR Interval: *Clinical Notes*

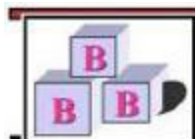
When the PR interval is long — We say there is **1st-degree AV block**. Given that the *isolated* finding of 1st degree AV block (*even if marked*) is usually *not* clinically significant (*Section 02.70*) — we generally **undercall** this finding. *Our* preference is to accept a PR interval = 0.21 second as normal (*and not to call 1st degree until the PR interval is ≥ 0.22 second = definitely more than 1 large box*). The PR interval in **Panel C** of **Figure 04.2-1** is clearly *more* than 1 large box in duration (*We estimate the PR interval in Panel C to be ~0.26 second*).

- We prefer not to use the term, “**borderline 1st degree**” — since all this really says, is that you *almost* have a finding that *even if present* would *not* be clinically important. Instead, *our* preference is to say the PR interval is either normal (≤ 0.21 second) or long (> 0.21 second).
- **Precise determination** of a PR interval that falls within the normal range is not necessary. Clinically — it *does not matter IF* the PR is 0.16, 0.17 or for that matter 0.19 second. Instead, it suffices to simply say — “*the PR interval is normal*”.
- **Norms for children** for the PR and QRS intervals are slightly different — because the pediatric heart is smaller than the adult heart. Specific normal limits for the various intervals are age-dependent. For example — a PR interval of 0.18 second may be prolonged for a *young* child, whereas it would be normal for a young adult. For the purposes of this ePub — it suffices to be aware that interval duration may differ slightly because of the smaller heart of a younger patient.

04.4 – Memory Aid: *How to Recall the 3 ECG Intervals*

An easy way to recall the *upper* limits of normal for the 3 ECG intervals is simply to *think of the numbers “1” and “2”* :

- The **PR interval** is **long** (*in adults*) — IF it is clearly *more* than “1” *large* box (>0.20 - 0.21 second in duration).
- The **QRS** is **long** — IF it measures *more* than “1/2” a *large* box (>0.10 second in duration).
- The **QT interval** is **long** — IF it comprises *more* than “1/2” the R-R interval.



Bundle Branch Block

05.1 – The QRS Interval: *What is Normal QRS Duration?*

The **QRS interval** represents the time it takes for ventricular depolarization to occur. With sinus rhythm in adults — the process of ventricular activation should **normally** be complete in **no more** than **0.10 second**. This means that IF the QRS complex is *longer* than **half a large box** in duration — that the QRS is “wide” (**Figure 05.5-1**):

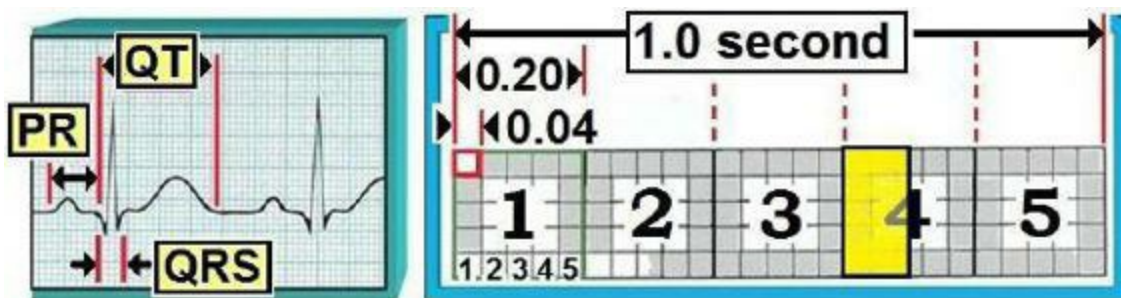


Figure 05.1-1: The QRS is “wide” — IF it measures *more* than 0.10 second (*which is more than half a large box in duration*).

Clinical Notes: QRS duration can be measured from *any* of the 12 leads of a standard ECG. **Select** the lead *in which* the QRS complex **appears** to be *longest*.

- Practically speaking — all that matters is whether the QRS is normal or wide. Precise measurement of QRS duration for a complex that is clearly *within* the normal range is *not* necessary. Given that 0.10 second is the *upper normal* limit for QRS duration in adults — the QRS is said to be “**wide**” — IF it measures **more** than **half a large box** in duration (**Figure 05.1-1**).
- These limits for QRS interval duration do *not* hold true for children (*for whom lesser degrees of QRS prolongation may be abnormal*).

05.2 – IF the QRS is Wide: *What Next?* (BBB Algorithm)

IF the QRS complex is wide — We suggest that you **STOP!** At this point —**Short-circuit** your *usual* sequence. Instead of your usual *systematic* approach — We suggest the following:

- First — **Ensure** that the **patient** is **stable!** This is because IF the patient is unstable (*and the rhythm is VT = Ventricular Tachycardia*) — you may need to *immediately* shock (*Section 02.47*).
- But — IF the QRS is wide and the rhythm is supraventricular — the **next step** in your ECG interpretation approach should be to proceed according to the **Algorithm** shown in **Figure 05.2-1**:

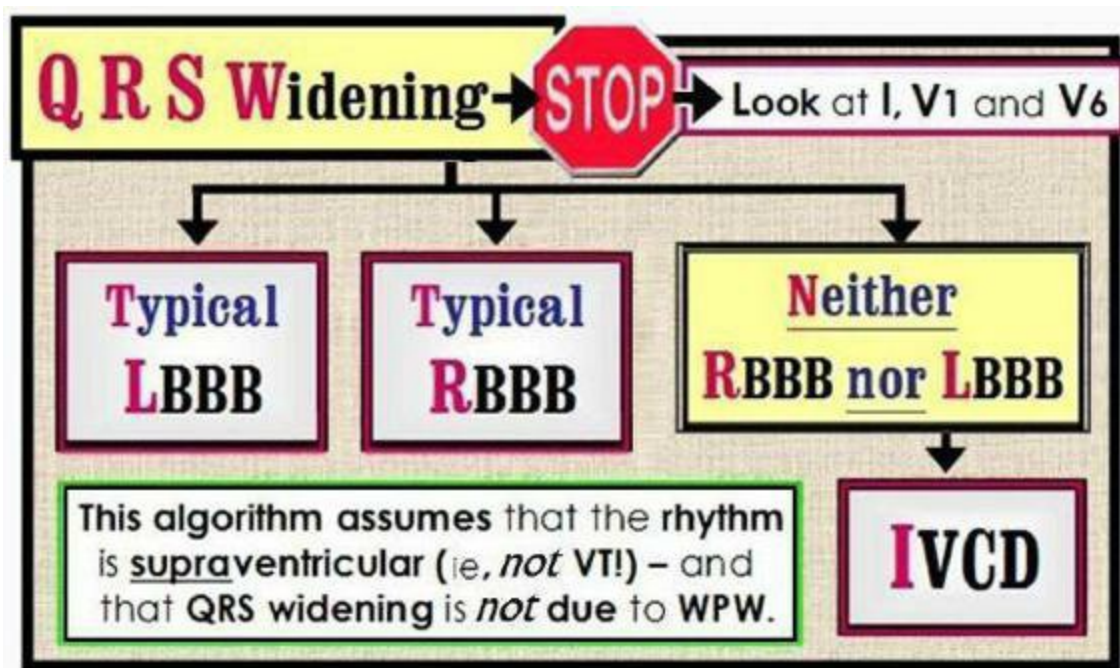


Figure 05.2-1: Algorithm — for how to proceed IF the rhythm is supraventricular (ie, *not WPW or VT*) and the QRS is wide. You should be able to determine the type of conduction defect in *no more* than a few seconds (*See text*).

KEY Point: — IF the QRS is Wide and the rhythm is supraventricular (ie, *sinus, AV nodal*) — then **determine *WHY*** the QRS is wide before proceeding further with your interpretation. Practically speaking (*and assuming we are not dealing with VT or WPW*) — there are only 3 reasons for QRS widening with sinus rhythm:

- There is **typical RBBB** (*Right Bundle Branch Block*).
- There is **typical LBBB** (*Left Bundle Branch Block*).
- The QRS is wide but neither typical RBBB nor typical LBBB is present. In this case, the reason the QRS is wide will be **IVCD** (*IntraVentricular Conduction Delay*).

The “good news” (*as discussed on the next few pages*) — is that use of the **Algorithm** in Figure 05.2-1 allows **accurate diagnosis** of the type of conduction defect (*BBB or IVCD*) in **less** than **5 seconds**!

- The reason we can *accurately* diagnose the type of conduction defect so quickly — is that: **i)** There are *only 3* possible answers (*typical RBBB; typical LBBB; or IVCD*); and ii) We **only need** to look at **3 leads** to make the diagnosis. **NOTE:** With experience — You’ll look at *all 12* leads for finer aspects of ECG interpretation. But only 3 leads are needed to diagnose the *type* of conduction defect.
- The **3 KEY leads** (*and the only 3 leads needed*) to determine the *type* of conduction defect (*RBBB, LBBB, or IVCD*) — are **leads I, V1, and V6**. **NOTE:** We key in on **2 left-sided leads** (*leads I, V6*) — and 1 right-sided lead (*lead V1*) when using Figure 05.2-1 to diagnose the *type* of conduction defect:

- **Remember** — Use of the algorithm in [Fig. 05.2-1](#) implies that the rhythm is supraventricular (and not VT or WPW).

05.3 – FIGURE 05.3-1: *Why the Need for the BBB Algorithm?*

The reason you want to recognize conduction defects before you get too far in the interpretation process — is that criteria for axis, LVH, RVH, ischemia and infarction are *all different* when there is BBB or IVCD! Consider the example in [Figure 05.3-1](#), obtained from a hemodynamically stable patient with a history of heart failure:

- Has this patient had an *anterior* MI (*slanted red arrows*) at some point in the past?
- Is there *ongoing* lateral ischemia (*red circle*) in lead V6?

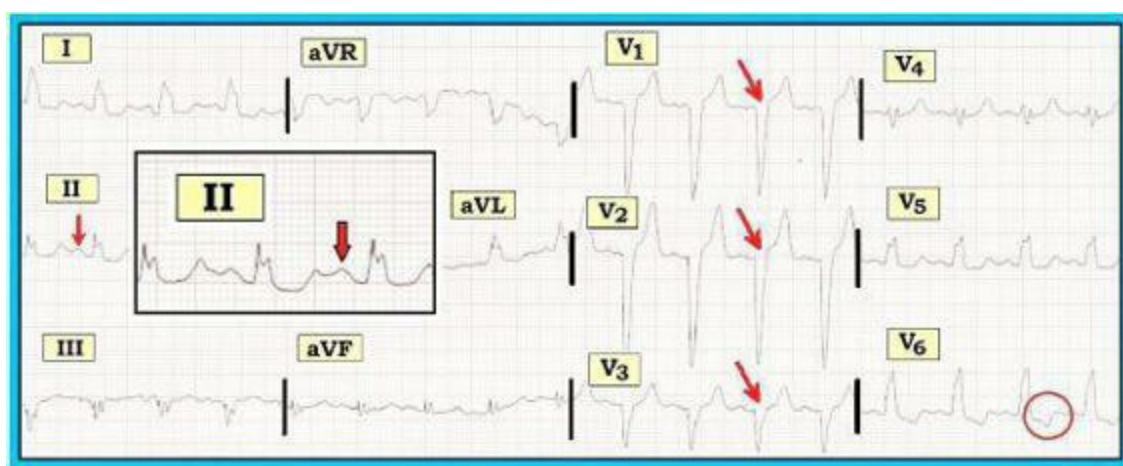


Figure 05.3-1: 12-lead ECG obtained from a stable patient with a history of heart failure. The rhythm is sinus (*vertical arrow in lead II insert*) — and the QRS is wide. *What next?* Has there been *anterior* infarction (*slanted arrows*)? Is there *lateral* ischemia (*red circle in lead V6*)?

Answer to Figure 05.3-1: The patient is hemodynamically stable. The rhythm is sinus (*vertical arrow in the lead II insert*) — and the QRS complex is wide. Thus, although one might be tempted to call *anterior* MI (*slanted arrows in V1, V2, V3*) and ongoing ischemia (*red circle in lead V6*) — the Algorithm in [Figure 05.2-1](#) advises us to first **STOP** and diagnose **WHY** the QRS is wide (?) before proceeding further.

- As we will discuss momentarily — the reason for QRS widening in [Figure 05.3-1](#) is **complete LBBB** (See Section 05.6). As a result — the deep *anterior* QS complexes and *lateral* ST-T wave changes are simply expected consequences of LBBB and *not* indicative of prior infarction or ischemia.
- **BOTTOM Line:** Had we *not* STOPPED at an *early* point in the interpretation process to assess the reason for QRS widening — we would have spent *needless* time thinking the patient had potentially active ischemia/infarction — when in reality there is *no* evidence of acute change on this ECG.

05.4 – Typical RBBB: Criteria for ECG Recognition

The 12-lead appearance of typical *complete* RBBB in the 3 *KEY* leads (*leads I, V1, V6*) is schematically shown in **Figure 05.4-1**. ECG criteria for diagnosis of *complete* RBBB include the following:

- **QRS widening** to *at least 0.11 second*.
- An **rSR'** or **rsR'** in *right-sided* lead **V1**.
- A **wide terminal S wave** in lead **I** and lead **V6**. The QRS complex is usually predominantly positive in these *left-sided* leads with RBBB. There may or may not be an initial small q wave. The *KEY* to *confirming* the diagnosis of RBBB is the *wide terminal S wave* in leads V1, V6 (**Figure 05.4-1**).

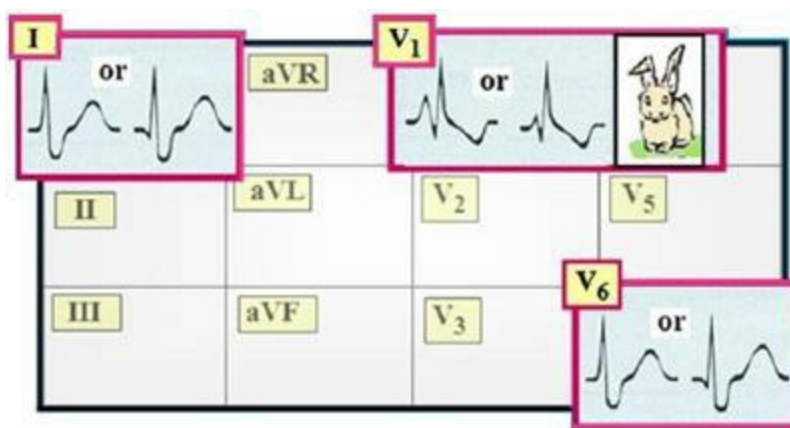


Figure 05.4-1: Schematic ECG of typical *complete* RBBB in the 3 *KEY* leads. Note the rsR' in lead V1 and wide terminal S waves in leads I, V6. **Memory Aid** — Think of **RBBB** and the "**R**'s" — in that there is an **rSR'** complex with a *taller Right rabbit ear* (the R') in a **Right-sided** lead (= lead V1).

Clinical Example of Complete RBBB: We illustrate application of the criteria for diagnosis of *complete* RBBB by our *sequential* approach to the 12-lead ECG shown in **Figure 05.4-2**:

- The rhythm in this ECG is **sinus tachycardia** at a rate just over 100/minute (*arrow in lead II indicating upright sinus P wave*).
- The **QRS** complex is **wide** (≥ 0.11 second). QRS duration is best assessed from a lead where the QRS is well defined and its duration appears to be longest. Despite seemingly normal QRS duration in several leads on this tracing (*leads aVL, V3, V4*) — there is *little* doubt that the QRS is clearly *more* than half a large box in duration in lead V1.
- Identification of a sinus rhythm *with* QRS widening is indication to **STOP** our systematic approach and branch to the **Algorithm** in **Figure 05.2-1**. We focus on the 3 *KEY* leads (*leads I, V1, V6*). As shown in the inserts in **Figure 05.4-2** — *complete* RBBB is diagnosed by the *typical* **rSR'** in V1 (*with taller right rabbit ear*) and the **wide terminal S wave** in leads I, V6.

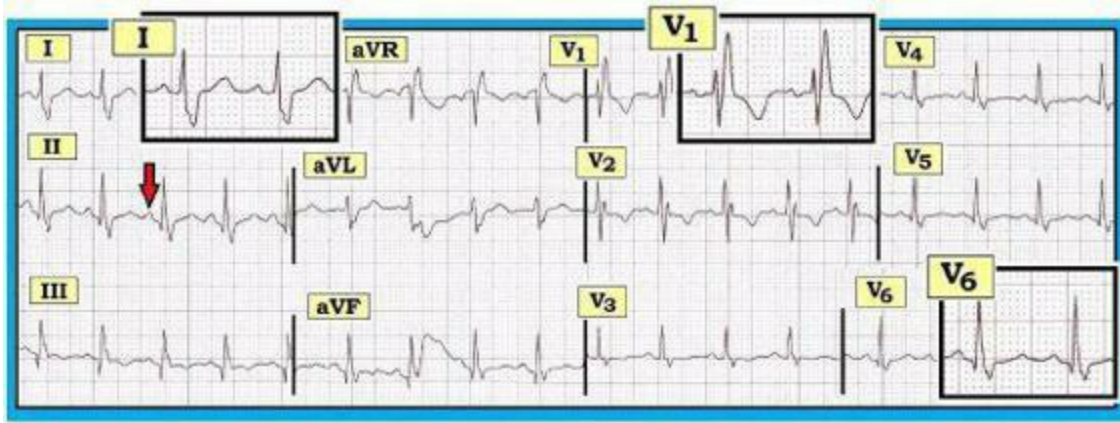


Figure 05.4-2: 12-lead ECG showing *complete RBBB* (See text).

05.5 – RBBB: Clinical Notes

The *upper normal* limit for QRS duration in adults is **0.10 second** (*half of a large box*). Anything *more* than 0.10 second is long (Section 05.1).

- **Complete RBBB** may occur with a **QRS** of **0.11 second**. This *differs* from the situation for LBBB — for which the QRS must attain ≥ 0.12 second (Section 05.6). The RV (*Right Ventricle*) is much *thinner* than the LV — so RBBB may be “*complete*” with a QRS complex that is *not* as wide as it needs to be when there is *complete* LBBB (*conduction through nonspecialized myocardial fibers in the blocked right ventricle may be complete within 0.11 second — whereas it will take ≥ 0.12 second to get through the thicker LV when there is LBBB*).
- An **incomplete RBBB** is said to be present — IF QRS morphology is typical in leads I,V1,V6 — but the QRS is *not* prolonged to at least 0.11 second (See Section 05.18).
- RBBB is a **terminal delay**. It does *not* affect the initial part of ventricular activation (*during which time the septum is activated and during which time Q waves are written*). As a result — **Q waves** may still be written *despite* RBBB. This differs from LBBB — which alters septal and initial vector activation (*thereby hiding most Q waves!* — See Sections 05.7, 05.8). Note that *despite* the **complete RBBB** present in Figure 05.4-2 — **inferior Q waves** (in leads II,III,aVF) are still seen (*which may be indicative of inferior infarction of uncertain age*).
- The **terminal delay** of RBBB may account for the fact that: **i)** the R' in V1 may be quite tall (*the LV has already depolarized — so the small RV is unopposed as it depolarizes*); and **ii)** the terminal S wave in leads V1,V6 is wide (*conduction through the blocked RV is slow due to the RBBB*).
- **RBBB** is a **relatively common** conduction defect that is *not* necessarily associated with underlying pathology (*especially if seen in an otherwise healthy adult with a normal physical exam and no history of heart disease*). IF doubt exists about possible *underlying* heart disease — an **Echo** could be obtained (*though an Echo is not necessarily needed on a routine basis*). In contrast — *new* RBBB in a patient with *acute* MI is clearly cause for concern (*suggests a larger infarct and more worrisome course*).
- In contrast to **RBBB** (*which may be a relatively benign finding in otherwise healthy adults*) — the finding of **complete LBBB** is *almost always* associated with some significant *underlying* cardiac pathology.

05.6 – Typical LBBB: Criteria for ECG Recognition

The 12-lead appearance of typical *complete* LBBB in the 3 *KEY* leads (leads I, V1, V6) is schematically shown in **Figure 05.6-1**. ECG criteria for *complete* LBBB include the following:

- **QRS widening** to at least **0.12 second** (whereas complete RBBB can occur with a $QRS = 0.11$ second).
- An **upright** (monophasic) QRS complex in leads **I** and **V6** that may (or may not) be notched. But — there should not normally be any q wave in *either* lead I or V6.
- A predominantly **negative** QRS complex in lead **V1**. There may (or may not) be an initial small r wave in lead V1 (lead V1 may show either a QS or rS complex).

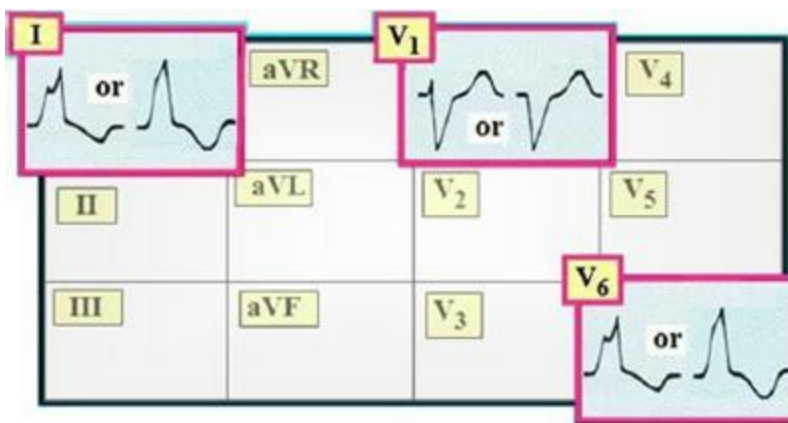


Figure 05.6-1: Schematic ECG of typical *complete* LBBB in the 3 *KEY* leads. NOTE: — there should never normally be a Q wave in a *left-sided* lead (lead I, V6) with typical LBBB unless there has been *prior* infarction (Section 05.28).

05.7 – FIGURE 05.7-1: LBBB alters Septal Activation

Normally — the very first part of the ventricles to depolarize (after the impulse passes through the AV node and Bundle of His) — is the **left side** of the **ventricular septum**. As a result — septal depolarization **normally** moves from **left-to-right** (arrow pointing left-to-right in **Panel A** of Figure 05.7-1).

- Since the **RBB** (Right Bundle Branch) travels down the *right* side of the septum — this initial **left-to-right** direction of septal depolarization does not change when there is **RBBB** because the *left* side of the septum *remains intact* and still initiates septal depolarization (**Panel B**).
- In contrast, with **LBBB** — the normal **left-to-right** direction of septal activation will change. This is because the block with LBBB prevents the initial vector of septal activation from starting on the left (**Panel C**). As a result — ventricular activation with **LBBB** moves almost entirely from **right-to-left**. Thus, the *initial* part of the QRS complex (during which time Q waves are written) is altered by LBBB that *changes* the direction of septal depolarization.

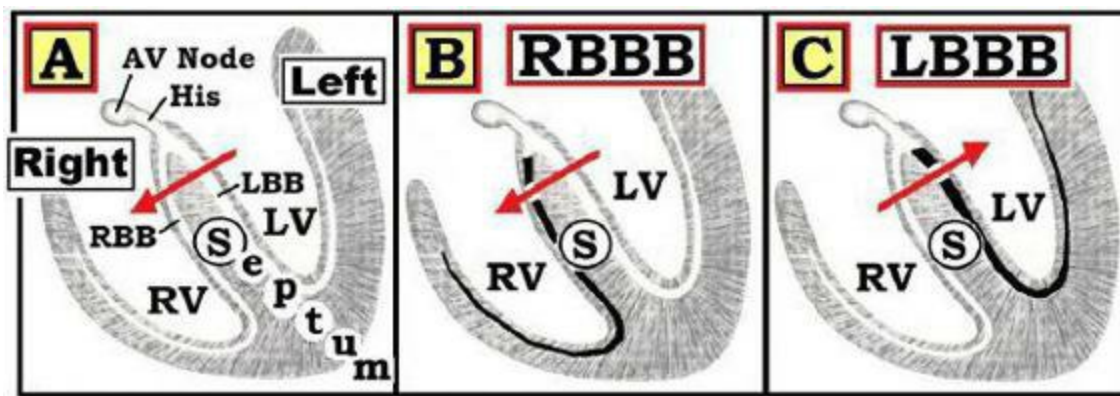


Figure 05.7-1: Sequence of normal septal activation — in which there is *left-to-right* septal depolarization (arrow in Panel A). This left-to-right direction is *preserved* with **RBBB** (Panel B) — but becomes *right-to-left* with **LBBB** (arrow in Panel C).

It is this *altered* direction of *initial* septal activation that accounts for many of the **ECG findings** of **LBBB**:

- Because *septal* activation with **LBBB** moves *right-to-left* — the small “*septal q waves*” that are often normally present in one or more *lateral leads* (*I, aVL, V6*) **should not be seen**. IF a Q wave (even a *small one*) is seen in a *lateral lead* (*I, aVL or V6*) in a patient with LBBB — that patient has had a *prior* infarction.
- Following *right-to-left* septal activation (arrow in Panel C of Figure 05.7-1) — ventricular activation continues to the *left* and *posteriorly*, as the large LV is depolarized. This results in the totally *upright* complex (*R wave*) seen in *left-sided leads* (*I, V6*) with LBBB. There may or may not be a notch = RR' in leads *I, V6* (Figure 05.6-1).
- *Right-to-left* septal activation, followed by continued leftward depolarization (as the rest of the LV is activated) — results in a predominantly *negative* complex (*QS or rS*) in *right-sided lead* *V1* with LBBB. Neighboring *anterior leads* (*V1, V2, V3*) often manifest a QS complex with LBBB without this indicating prior MI (Figure 05.8-1 in the next Section).

05.8 – FIGURE 05.8-1: Clinical Example of Complete LBBB

We illustrate application of the criteria for diagnosis of *complete* LBBB by our *sequential* approach to the 12-lead ECG shown in Figure 05.8-1:

- The rhythm in this ECG is **sinus tachycardia** at a rate of ~100/minute (arrow in lead *II* indicating *upright sinus P wave*).
- The **QRS** complex is **wide** (at least 0.12 second in duration).
- Identification of a sinus rhythm with QRS widening is indication to **STOP** our systematic approach and branch to the **Algorithm** in Figure 05.2-1. We focus on the **3 KEY leads** (leads *I, V1, V6*). As shown in the inserts in Figure 05.8-1 — **complete LBBB** is diagnosed by the predominantly *negative* QRS complex in lead *V1* and the monophasic *upright R wave* in leads *I* and *V6*.
- Note that the R wave is *notched* in lead *I* of Fig. 05.8-1 — but not in lead *V6*. R wave notching in one or more lateral leads may or may not be seen with LBBB. No definite clinical

implications are conveyed by its presence or absence.

- Note also that there are **QS complexes** in leads **V1-thru-V3** and there is **ST-T wave depression** in several *lateral leads* (*I, V5, V6*). Both of these findings are common with LBBB, and do *not* convey any information regarding possible ischemia/infarction. In addition — the slight (*but not marked*) ST elevation seen in anterior precordial leads is common and an *expected* finding with LBBB.
- *Beyond-the-Core:* We also draw attention to the QS complex seen in leads III and aVF of Figure 05.8-1. Because LBBB changes the sequence of *initial* ventricular activation (*during which time Q waves are written*) — the presence of Q waves in leads *other than* the lateral leads is *not* indicative of infarction when there is LBBB. **Bottom Line:** All we can say about the ECG in Figure 05.8-1 is that there is *complete* LBBB. There is *no* evidence of any acute change.

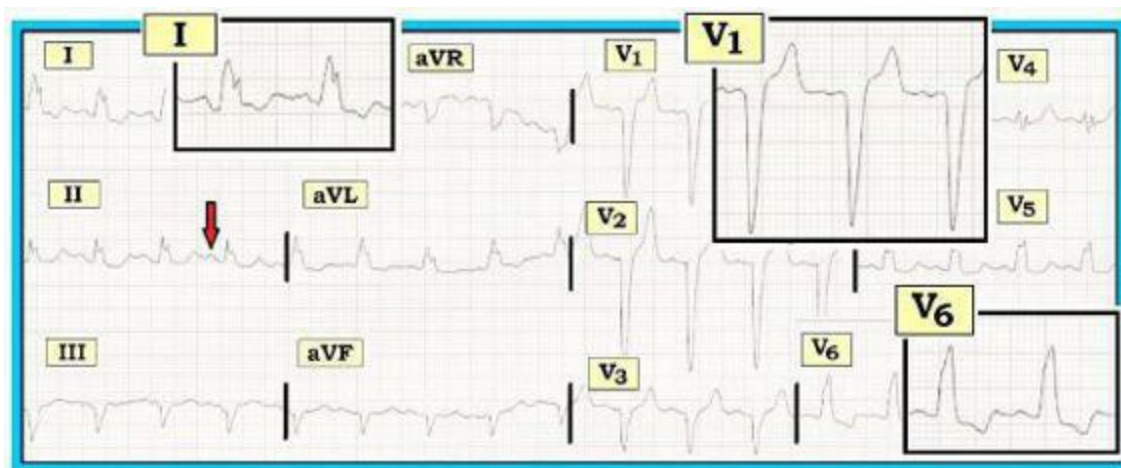


Figure 05.8-1: 12-lead ECG showing *complete* LBBB (See text).

05.9 – LBBB: Clinical Notes

The ECG finding of **LBBB** is virtually *always* associated with significant **underlying heart disease** (ie, *cardiomyopathy, longstanding hypertension with LVH, heart failure, coronary artery disease, valvular or congenital heart disease*). It is rare in adults (*or in children*) to find *isolated* “benign” LBBB.

- The **diagnosis** of *other conditions* (*ischemia, infarction, ventricular hypertrophy*) — will always be **more difficult** when there is **BBB**. This is *especially* true for LBBB — which changes the *direction* of *initial* septal activation (*Section 05.7*). That said — diagnosis of *other* conditions is *not* impossible on certain occasions (*See Sections 05.24-through-05.32*).
- The reason *no* “septal” q wave should be seen with **typical LBBB** in *either* lead **I** or lead **V6** — is that LBBB causes the septum to be activated from *right-to-left*. As a result — *left-sided* leads (*I, V6 and for that matter aVL and V5*) see the *initial* activation vector as coming *toward* instead of moving away from the left (**Panel C** in Figure 05.7-1). **IF** on the other hand, there has been **septal MI** — you may then see a small q wave in one or more lateral leads *despite* the LBBB.

05.10 – *Incomplete LBBB: Does it Exist?*

There is an entity known as “*incomplete LBBB*”. That said — *incomplete LBBB* is very uncommon, difficult to diagnose, and usually not clinically important to distinguish from *nonspecific IVCD*. This differs from the situation with *incomplete RBBB* which is very common and *easy* to diagnose (See Section 05.18).

- Complicating **diagnosis** of *incomplete LBBB* is uncertainty about when LBBB is *truly* complete. If for example, the QRS *continues* to widen over a period of months-to-years (say, from 0.12 to 0.14 to 0.16 second) — at what point was the LBBB “complete”? Recognition of *incomplete LBBB* is also made more difficult by the fact that *other* conditions (ie, *LAHB*, *LVH*) may slightly widen the QRS in a *similar-appearing* pattern.
- Practically Speaking — the diagnosis of *incomplete LBBB* is really a *retrospective* one. It is only *over time* that you can know with certainty that a *prior* ECG with *some* QRS widening was in fact one stage in an *evolving* process that ultimately led to *more* QRS widening that *finally* became *complete LBBB*. **BOTTOM Line:** While nice to be aware of the controversy and *lack* of consensus regarding the “definition” of *incomplete LBBB* — this diagnosis is difficult to prove and usually of *little* clinical benefit. We therefore suggest not to worry about trying to distinguish between “*incomplete LBBB*” vs *slight* QRS widening due to a *nonspecific IVCD* pattern.

05.11 – *IVCD: Criteria for ECG Recognition*

The ECG appearance of **IVCD** (*IntraVentricular Conduction Delay* or *Defect*) — is difficult to characterize. This is because IVCD is often the **end result** of a number of *different* pathophysiologic processes — rather than a discrete defect in the conduction system (*as usually occurs with RBBB or LBBB*).

- Examples of conditions that may lead to IVCD include myocardial infarction, cardiomyopathy with ventricular fibrosis, chamber enlargement — and/or any *combination* of these (*with or without an associated component of BBB*).

A *schematic* example of IVCD is shown in **Figure 05.11-1**. ECG diagnostic criteria for **IVCD** include the following:

- **QRS widening** to *at least 0.11 second*.
- Neither **typical RBBB** *nor* **typical LBBB** is present.

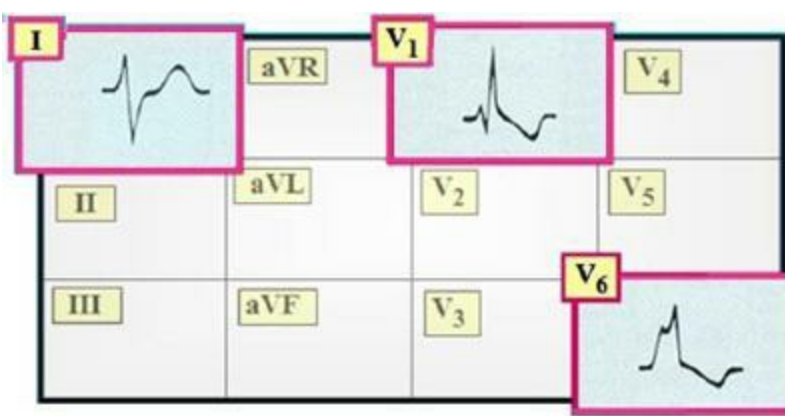


Figure 05.11-1: Schematic example of IVCD. QRS morphology is not typical here for either RBBB or LBBB in *all* 3 of the *KEY* leads (leads I and V1 are consistent with RBBB — but lead V6 suggests LBBB). Clearly — *many forms* of IVCD are possible in addition to the one shown here.

05.12 – IVCD: Clinical Notes

The term “**IVCD**” is used to describe the entity in which there is sinus rhythm with QRS widening — but QRS morphology in the 3 *KEY* leads (I, V1, V6) is not typical for either RBBB or LBBB.

- This use of the term “**IVCD**” is “***magic***” — as it tremendously simplifies (*and expedites*) the diagnostic process. Once we *rule out* VT and WPW — We know QRS widening in a supraventricular rhythm will be due to either RBBB, LBBB or IVCD (See **Algorithm** in **Figure 05.2-1**).
- IF criteria for RBBB and LBBB are not met (*in each of the 3 KEY leads*) — then **IVCD** is the reason for the wide QRS. *End of process!* We illustrate this concept in the clinical example of IVCD shown below (Figure 05.13-1).

NOTE: It will often be difficult (*if not impossible*) with IVCD to assess for LVH/RVH; *prior* MI; or *acute* ST-T wave changes.

- As is the case for RBBB and LBBB — the **clinical significance** of **IVCD** depends greatly on the setting in which it occurs. Slight QRS widening (*of up to 0.10-to-0.11 second*) — is occasionally seen in otherwise *healthy* young adults (*in which case, this slight QRS widening of “IVCD” is most often of no clinical significance*).
- IF doubt exists about the presence of underlying structural heart disease — an **Echo** could be obtained (*although an Echo is not necessarily needed on a routine basis*).
- In contrast — IVCD in an older adult with chest pain or heart failure may carry similar implications as would LBBB (ie, *coronary artery disease, infarction, cardiomyopathy*). **Clinical correlation is everything!**

05.13 – FIGURE 05.13-1: Clinical Example of IVCD

We illustrate application of the criteria for diagnosis of IVCD by our *sequential* approach to the 12-lead ECG shown in **Figure 05.13-1**:

- The rhythm in this ECG is **AFib** (*Atrial Fibrillation*) — because it is *irregularly* irregular

without P waves.

- The **QRS** complex is **wide** (at least 0.12 second).
- Identification of a supraventricular rhythm with QRS widening is indication to **STOP** our systematic approach and branch to the **Algorithm** in **Figure 05.2-1**. We focus on the **3 KEY leads** (leads I, V1, V6). As shown in the inserts in **Figure 05.13-1** — **IVCD** is diagnosed because QRS morphology does not resemble either RBBB or LBBB in *each* of the 3 key leads. That is, the QRS complex in lead I of **Fig. 05.13-1** looks like LBBB — but not in leads V1, V6 (*which if anything look more like RBBB*). **Bottom Line:** One can't say much else about this tracing beyond AFib with a *controlled* ventricular response plus QRS widening due to IVCD.

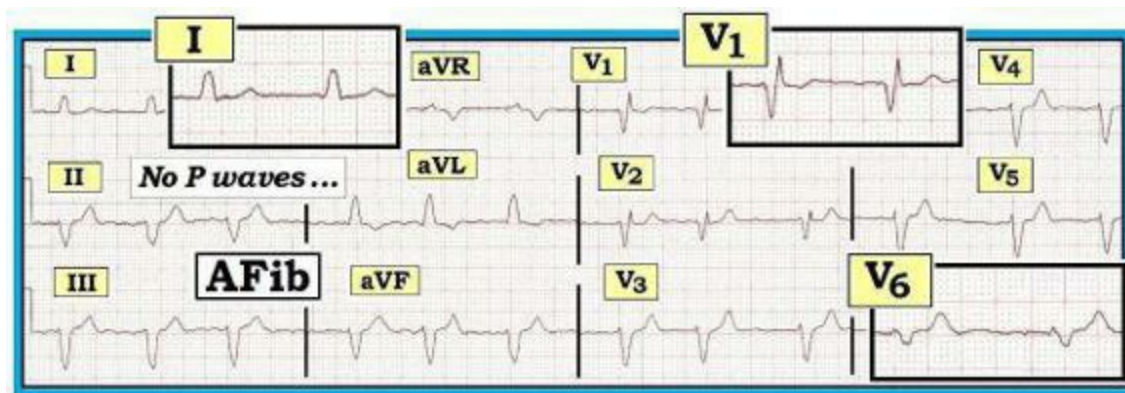


Figure 05.13-1: 12-lead ECG showing IVCD (See text).

05.14 – ST-T Wave Changes: *What Happens with BBB?*

RBBB and LBBB each alter the *sequence* of ventricular depolarization. This is why they produce the alterations in QRS morphology discussed in this Section. As a *direct* result of this altered sequence of activation — these conduction defects *also* alter the sequence of ventricular *repolarization*, which leads to development of **secondary** (2°) **ST-T wave changes** (**Figure 05.14-1**).

- The ST-T wave changes shown in **Fig. 05.14-1** are called *secondary* — because they are the *direct result* of (ie, *they are secondary to*) the conduction defect itself. That is — We *expect* to see this pattern of ST-T wave response shown in **Figure 05.14-1** as a normal part of RBBB or LBBB.

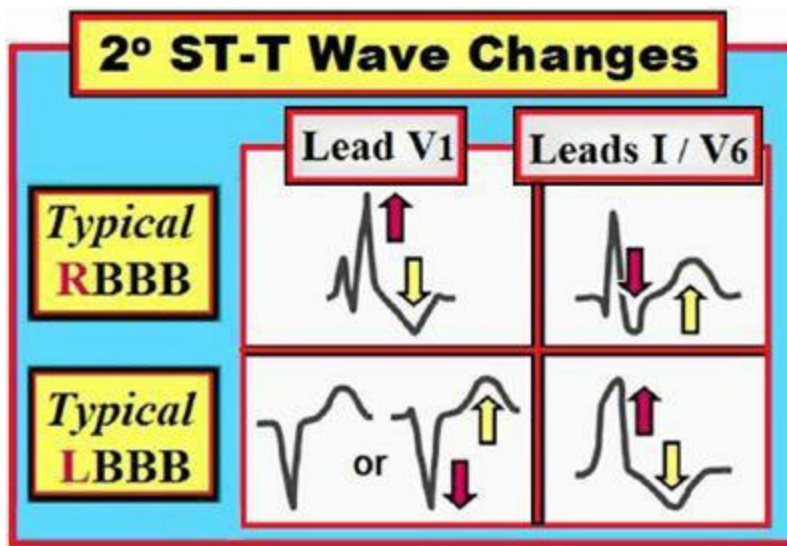


Figure 05.14-1: The *expected* ST-T wave response to either *typical* RBBB or *typical* LBBB is “**ST opposition**”. That is — the ST segment *and* T wave should be ***oppositely directed*** to the ***last QRS deflection*** (red and yellow arrows) in *each* of the 3 **KEY** leads (= *leads I, V1, V6*). Deviation from this pattern is abnormal — and indicates a ***primary*** (1°) **ST-T wave change** (*that suggests ischemia or infarction*).

KEY Points: The *beauty* of the “**ST opposition**” rule in **Figure 05.14-1** — is that it allows you to assess ST-T morphology with BBB *within* 2-3 seconds!

- Although we do look at *all* 12 leads with BBB, when applying the ST-T wave *opposition* principle — Look *only* in the 3 **KEY** leads (= *leads I, V1, V6*). Once you have done so — You can refine your interpretation by looking at ST-T wave changes in the remaining leads (*See Section 05.15*).
- **NOTE:** The “**ST opposition**” rule works for RBBB and LBBB — but *not* for IVCD (*which is why it is often quite difficult to assess for acute changes with IVCD*).

05.15 – FIGURE 05.15-1: *Assessing ST-T Wave Changes with BBB*

Apply the “**ST opposition**” rule to the examples of RBBB and LBBB shown in **Figure 05.15-1** (*in which we reproduce the ECGs previously shown in Figure 05.4-2 and Figure 05.8-1*). Are the ST-T waves in the 3 **KEY** leads doing what they should for bundle branch block seen in each case?

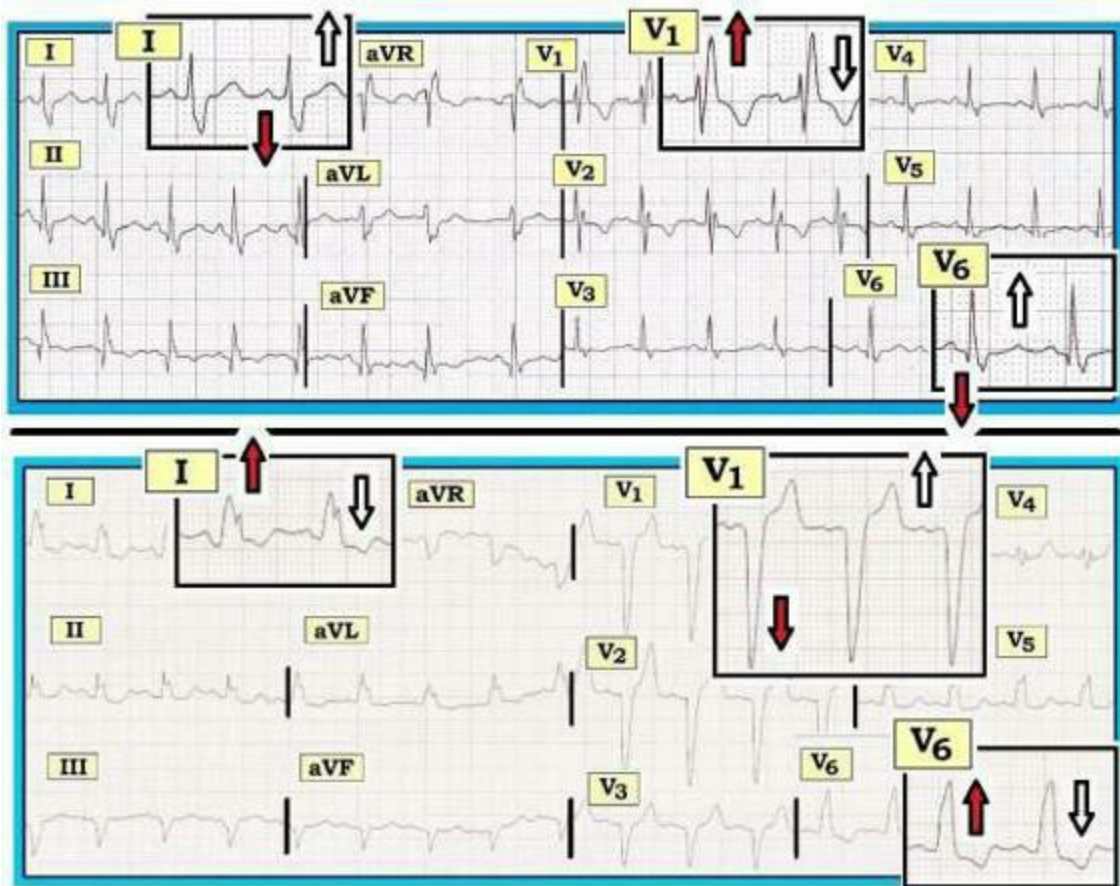


Figure 05.15-1: Are ST-T changes *as expected* for the examples of **typical RBBB** (Top tracing) and **LBBB** (Bottom tracing) seen here?

Answer to Figure 05.15-1: Sinus rhythm with QRS widening is seen for both tracings shown here. Focus on the **3 KEY leads** (I,V1,V6) allows *rapid* diagnosis of **complete RBBB** (top tracing) and **complete LBBB** (bottom tracing). It should now take *no more* than seconds to establish that ST-T waves (white arrows) are **oppositely directed** to the **last QRS deflection** (red arrows) in each of the 3 key leads. Looking at this in more detail:

- **TOP Tracing** — Recognition of the rSR' (with taller right rabbit ear) in V1 and wide terminal S waves in leads I,V6 define the conduction defect as **RBBB**. The last QRS deflection in lead V1 is upward (the R' = red arrow in V1) — so the ST-T wave is *appropriately* negative (white arrow). Terminal S waves in leads I,V6 inscribe a downward deflection (red arrows in these leads) — so the T wave is *appropriately* upright in both lead I and V6 (white arrows).
- **BOTTOM Tracing** — Recognition of a monophasic upright R wave in left-sided leads I,V6 and a predominantly negative QRS complex in lead V1 defines the conduction defect as **LBBB**. The *only* QRS deflection in leads I,V6 is upward (the monophasic R wave = red arrows in I,V6) — so the ST-T wave is negative in these leads (white arrows). Lead V1 manifests a predominantly negative QRS deflection (red arrow in V1) — so the ST-T wave is *appropriately* upright in this lead (white arrow).
- **NOTE:** Neighboring leads may or may not manifest **ST-T wave opposition** to the last QRS deflection. For example, there *is* ST-T wave opposition for *anterior* leads V2,V3 in the *bottom* tracing (negative QRS with upright T wave) — but *not* necessarily for all of the other leads. For this reason — Our focus is on looking at the **3 KEY leads** = leads I, V1 and V6.

05.16 – RBBB *Equivalent* Patterns

The shape of the QRS complex in lead V1 may vary greatly with RBBB. It will not always show a "neat" rSR' (or rsR') in this lead. Instead, any of the patterns shown for lead V1 in **Figure 05.16-1** qualify for diagnosis of **RBBB** — as long as the QRS complex is widened (to ≥ 0.11 second) — and as long as there is a wide *terminal* S wave in *left-sided* leads *I* and *V6*.

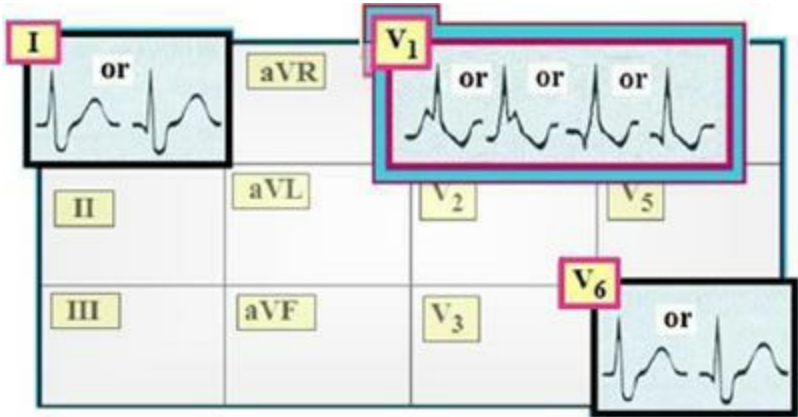


Figure 05.16-1: RBBB “*Equivalent*” Patterns in lead V1. Despite *lack* of a *taller-right-rabbit-ear* rSR’ pattern in lead V1 — **RBBB** is *still* present IF: **i)** the QRS is wide (≥ 0.11 second); **ii)** an *upright* QRS complex *similar* to one of the patterns shown here is seen in lead V1; and **iii)** a wide *terminal* S wave is seen in lead I and lead V6.

KEY Points: Normally — the QRS complex in lead V1 is *predominantly* negative. This is because this *right-sided* lead (*V1*) sees electrical activity as *moving away* from V1 (or *toward the left ventricle*). The finding of predominant *positive* activity (ie, a *tall R wave*) in *right-sided* lead V1 is not “normal”.

- IF the rhythm is sinus and the QRS is wide with an “**RBBB-*Equivalent*”** pattern in lead **V1** — then the conduction defect is **RBBB** — as long as there are **wide terminal S waves** in leads **I** and **V6** (Figure 05.16-1).
- Reasons why the typical triphasic (*rsR'*; *taller right rabbit ear*) complex in V1 may be *lost* in some patients with RBBB include scarring (*as may occur with cardiomyopathy*) and/or infarction (*which would be especially suggested by a qR pattern in lead V1*).

05.17 – FIGURE 05.17-1: *Is this RBBB?*

The schematic tracing in Figure 05.17-1 shows sinus rhythm and QRS widening.

- What *type* of BBB is seen in Figure 05.17-1?

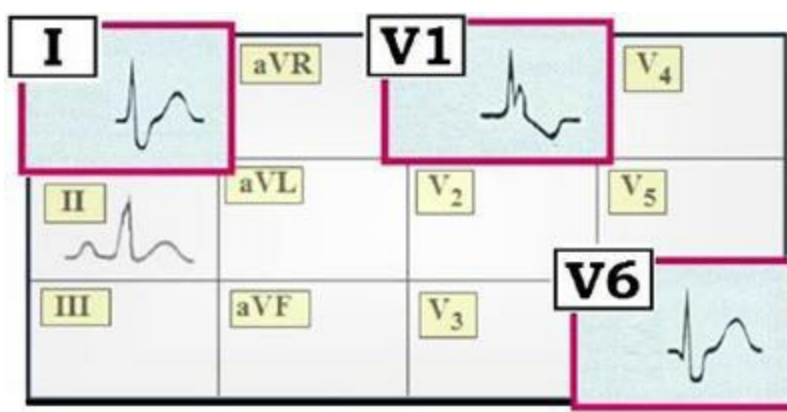


Figure 05.17-1: What type of conduction block is present?

Answer to Figure 05.17-1: Lead II shows the rhythm is sinus. The QRS is wide and upright in lead V1 — but not in the usual **rSR'** (taller right rabbit ear) pattern for typical RBBB. Instead, the left rabbit ear is taller. This qualifies as an **“RBBB Equivalent”** pattern (Figure 05.16-1). Since wide terminal S waves are seen in the lateral leads (I, V6) — this tracing satisfies criteria for **RBBB**. ST-T wave changes are appropriate (opposite the last QRS deflection in the 3 key leads) — so there are no acute changes.

05.18 – Incomplete RBBB: How is it Diagnosed?

As discussed in Section 05.5 — **IRBBB** (Incomplete Right Bundle Branch Block) is diagnosed when QRS morphology is consistent with RBBB (Section 05.4) but **QRS duration** is **less** than **0.11 second** (Figure 05.18-1):

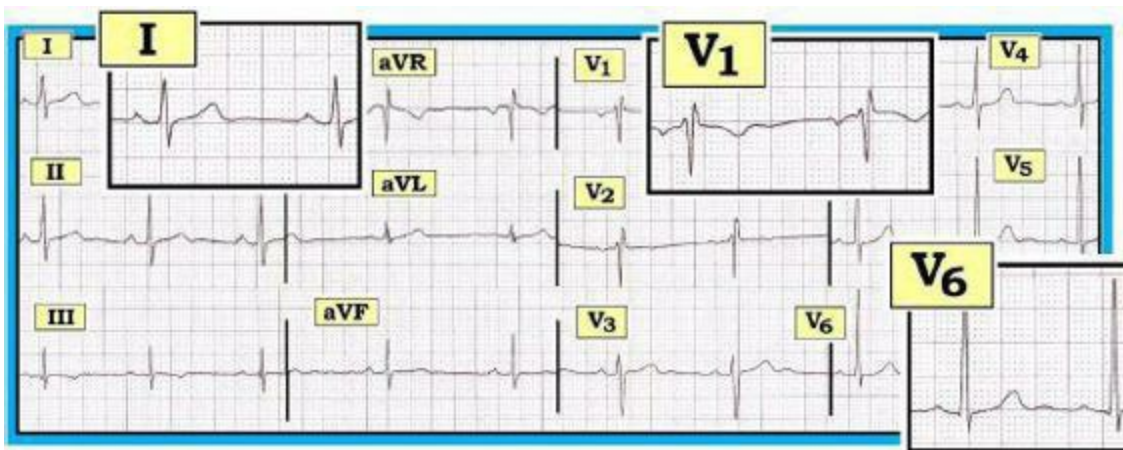


Figure 05.18-1: ECG showing **IRBBB** (Incomplete Right Bundle Branch Block) — obtained from a healthy young adult. An **rSr'** is seen in lead V1 with S waves in leads I, V6 — but QRS duration is 0.11 sec (not more than half a large box). Note that the S waves seen in leads I, V6 are narrow.

KEY Points about IRBBB: Patients with IRBBB run the gamut from *common* occurrence in *up to* ~5% of otherwise healthy young adults (*in which case it is benign*) — to IRBBB serving as an ECG sign of RVH/COPD; acute pulmonary embolus; or acute conduction system damage seen with acute MI. **Clinical correlation is everything!**

- An **rSr'** in lead **V1** (*and/or* in leads **III** or **V2**) — is a common *benign* finding in healthy young adults (*due to normal late depolarization of the RV outflow track*). IF, as is seen in Figure 05.18-1 there is *also* an S wave in leads **I** and **V6** — then we define this as **“IRBBB”**. If S

waves are missing — We simply say, “*an rSr’ is present in lead V1*”.

- Note that the ST-T wave in lead V1 of Figure 05.18-1 is inverted. This is an *expected* finding given the IRBBB pattern in V1 (*See Section 05.14 on the ST opposition rule*).
- *Beyond-the-Core*: Be aware that placing chest leads V1 and V2 an interspace *too high* is a technical mishap that may misleadingly induce an rSr’ pattern in these leads (*Section 03.21*).



PRACTICE – BBB:

Interpret the following 2 *schematic* and 2 *real* tracings (Sections 05.20-thru-05.23). Sinus rhythm is present in *each* tracing — but the QRS is wide. What *type* of conduction defect is present? Is there indication of possible *ongoing* ischemia/infarction in *any* of these tracings?

- Our *Answers* follow each case.
- **Hint:** Feel free to *refer back* to Figures 05.2-1; 05.4-1; 05.6-1; 05.11-1; and 05.14-1 while formulating your answers.

05.20 – PRACTICE: *Tracing A*

In *schematic Tracing A* (Figure 05.20-1) — there is sinus rhythm with QRS widening.

- What type of BBB is present? Is *anything else* going on?

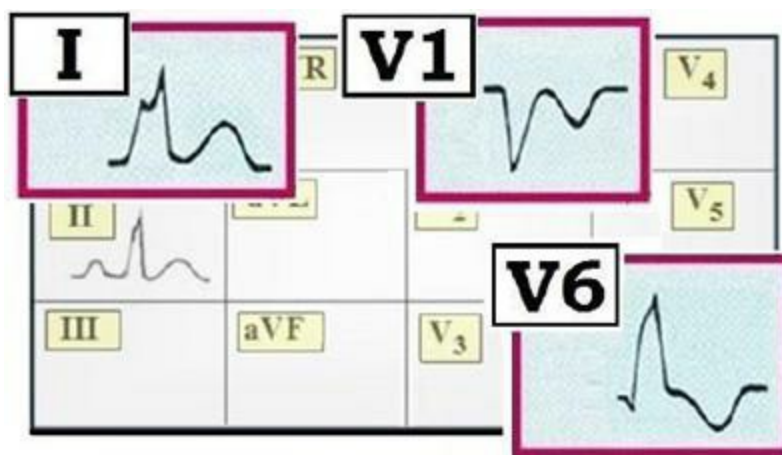


Figure 05.20-1: Practice Tracing A. What type of BBB is present?

ANSWER to Tracing A: There is sinus rhythm with QRS widening. QRS morphology is consistent with the pattern of **complete LBBB** in *each* of the 3 key leads (Section 05.6). However, a **Q wave** is present in Lead V6. This is not normally seen with *typical* LBBB. The finding of a Q wave in a *lateral* lead (I, aVL, or V6) with LBBB indicates *prior* infarction.

- There are also **abnormal** (ie, *primary*) **ST-T wave changes** in the 3 key leads As emphasized in Section 05.14 (Figure 05.14-1) — the T wave with *typical* LBBB should not be upright in lead I. The T wave should also not be negative in lead V1 with *uncomplicated* LBBB.
- **Bottom Line:** We interpret *schematic Tracing A* as showing **LBBB** with **primary ST-T wave changes**. This suggests infarction has taken place at some point in the past. Clinical history and comparison with prior tracings would be needed to determine if the changes seen are new, recent or old.

- *Beyond-the-Core*: Rather than “lateral” infarction — the Q wave in lead V6 indicates prior *septal* infarction. As discussed and illustrated in Section 05.7 — LBBB changes the direction of septal activation (*from left-to-right to right-to-left*). Thus, development of a Q wave in a *lateral* lead with LBBB indicates further disturbance with the process *septal* activation, presumably due to *septal* infarction.

05.21 – PRACTICE: *Tracing B*

In *schematic Tracing B* (Figure 05.21-1) — there is sinus rhythm with QRS widening.

- What *type* of conduction defect is present?

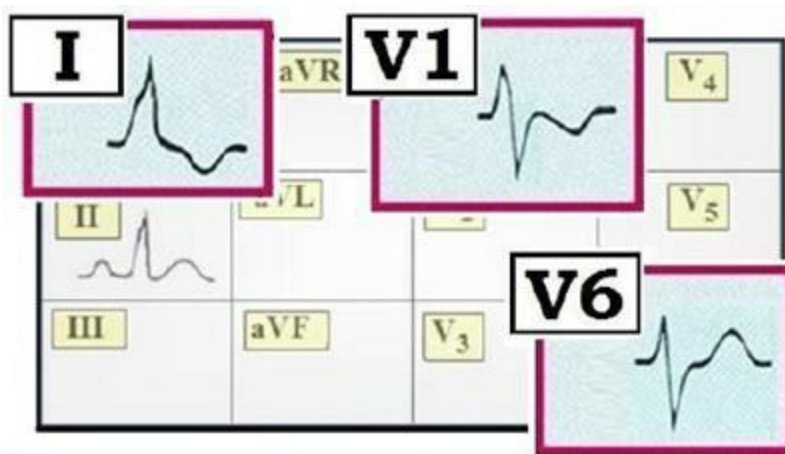


Figure 05.21-1: *Practice Tracing B.* What type of conduction defect is present?

ANSWER to Tracing B: There is sinus rhythm with QRS widening. That said — QRS morphology is not consistent with either RBBB or LBBB in *each* of the 3 key leads. That is — the monophasic R wave in lead I looks like LBBB; the wide terminal S wave in V6 is consistent with RBBB; and the R=S pattern in V1 looks like *neither* LBBB *nor* RBBB.

- By the process of elimination — we interpret the conduction defect in Figure 05.21-1 as **IVCD** (*Section 05.11*).
- ST-T wave morphology is difficult to interpret with IVCD. This is because the **ST Opposition Rule** does not necessarily work with **IVCD** (*Section 05.14*). As a result — ST-T waves are not necessarily directed opposite to the last QRS deflection in the 3 key leads. It may therefore not be possible to assess for ischemia/infarction when there is IVCD (*unless ST-T wave changes are extreme*).

05.22 – PRACTICE: *Tracing C*

There is again sinus rhythm with QRS widening in **Tracing C** (Figure 05.22-1).

- What type of BBB is present? Is *anything else* going on?

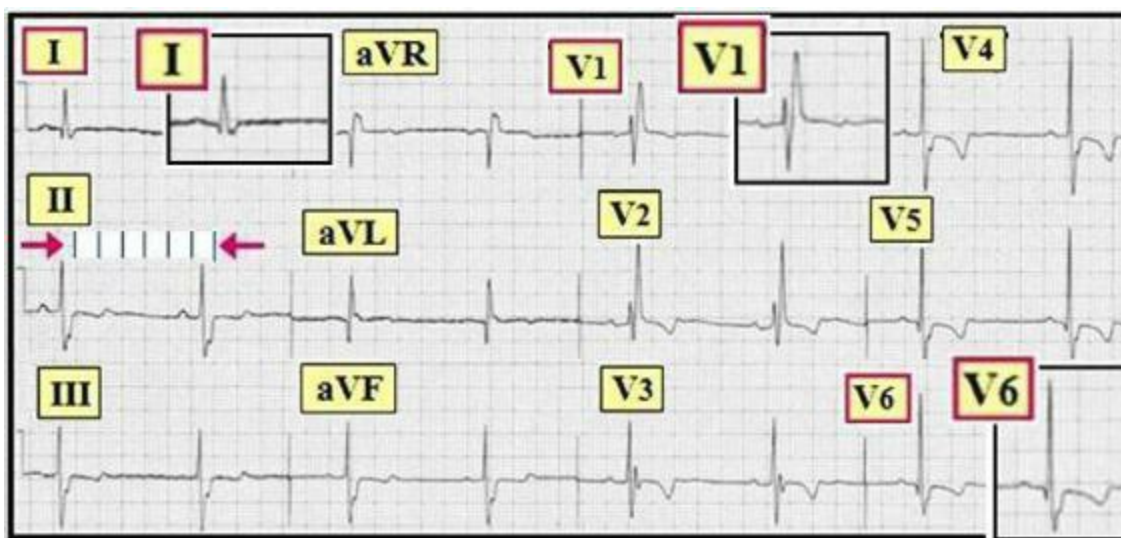


Figure 05.22-1: Practice **Tracing C**. What type of BBB is present? Is *anything else* going on?

ANSWER to Tracing C: The rhythm is sinus bradycardia at ~50/minute. The QRS complex is wide in a pattern consistent with **complete RBBB** (Section 05.4). That is — there is an rSR' (with taller right rabbit ear) in lead V1 with wide terminal S waves in *both* leads I and V6.

- **NOTE-1:** ST-T waves are not as expected for *uncomplicated* BBB (Section 05.14). That is — **ST-T waves** in **Figure 05.22-1** are **not oppositely directed** to the last QRS deflection in *each* of the 3 **KEY** leads. Instead — there is ST flattening in leads I, V1 and T wave inversion (*instead of an upright T wave*) in lead V6. Thus there are **primary ST-T wave changes** on this tracing — which suggests possible recent or *ongoing* ischemia/infarction.
- **NOTE-2:** The ECG in **Figure 05.22-1** — provides an excellent example of how we use the concept of **neighboring leads** to refine our interpretation. Thus, the **ST Opposition Rule** (**Figure 05.14-1**) — allows us to quickly determine that ST-T wave depression in lead V6 is not normal for *typical* RBBB. We therefore should not expect neighboring leads (ie, *leads V4, V5*) to manifest the deep *symmetric* T wave inversion that is seen in **Fig. 05.14-1**. This suggests that in addition to RBBB — there is a wider area of *lateral* ischemia.
- Finally — there are relatively *larger-than-expected* **Q waves** in the **lateral leads** (*leads I, V5, V6, and especially aVL*). Especially in the context of RBBB and *primary* ST-T wave changes — these Q waves (*particularly the relatively deep Q in lead aVL*) may well reflect infarction of *uncertain* age.
- **Bottom Line:** — We interpret the ECG in **Tracing C** as showing sinus bradycardia with *complete* RBBB. There are *lateral* Q waves of *uncertain* significance and *primary* ST-T wave changes that suggest **possible infarction/ischemia** of **uncertain** age *in addition to* RBBB. This *could be* acute. *Clinical correlation* is needed.
- **Beyond-the-Core:** The question often arises as to *how far over* in the anterior leads ST-T wave

depression from RBBB may be seen? We *expect* to see ST-T wave depression in leads V1,V2 with RBBB as part of the normal secondary ST-T wave changes of BBB. This was seen in the 12-lead ECG example of *typical* RBBB shown in Figure 05.4-2. Sometimes *anterior* ST-T depression with typical RBBB may carry over as far as lead V3 — but it should usually *not* go beyond this! It should be clear that ST-T wave appearance in Figure 05.22-1 is *not* normal because: **i)** the ST-T wave should be upright in lead V6 (*as well as in leads V4,V5*); and **ii)** the ST segment becomes *coved* in lead V3 — and the *amount* of T wave inversion in V3 is *increased* compared to V2 (*whereas with simple RBBB we would expect a decrease in amount of ST-T wave change from V2-to-V3*).

05.23 – PRACTICE: *Tracing D*

A final example of sinus rhythm with QRS widening is seen in **Tracing D** (Figure 05.23-1).

- What *type* of conduction defect is present?

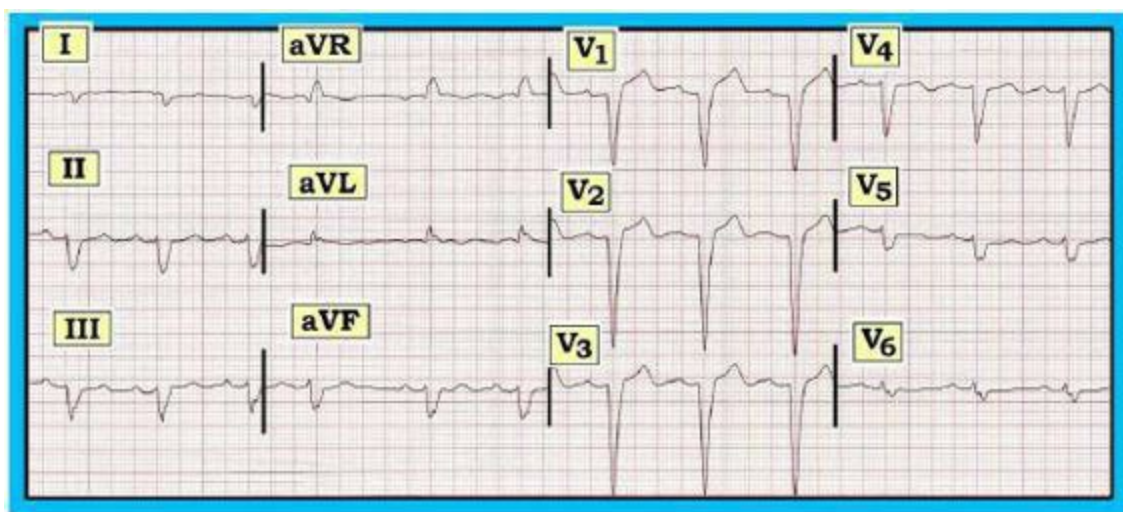


Figure 05.23-1: *Practice Tracing D. What type of conduction defect is present?*

ANSWER to Tracing D: There is sinus rhythm with QRS widening. There is a PAC (*the 4 beat occurs early*). QRS morphology is not consistent with either RBBB or LBBB in *each* of the 3 key leads. That is — the *negative* QS complex in lead V1 is consistent with LBBB — but, if anything the wide terminal S waves in the tiny complexes seen in leads I and V6 are consistent with RBBB.

- By the process of elimination — we interpret the conduction defect in Figure 05.23-1 as **IVCD** (*Section 05.11*).
- As stated — ST–T wave morphology is difficult to interpret with IVCD because the ST *Opposition* Rule is not necessarily followed (*Section 05.14*). We would interpret ST–T wave changes seen here as “nonspecific”. Clinical correlation is needed to determine the likely significance of the conduction defect seen.

05.24 – Diagnosing BBB + Acute MI



Diagnosis of ischemia/infarction is *always* more challenging when there is *underlying* BBB (*Bundle Branch Block*). That said — suspicion of old or acute events will *surprisingly often* be possible.

- The goal of Sections 05.24-thru-05.29 — is to briefly review key considerations for assessing the patient with a conduction defect for possible ischemia/infarction.

05.25 – Begin with the ST Opposition Rule

While *not* infallible, the ***ST opposition rule*** (Sections 05.14, 05.15) — is a good place to start when assessing patients with RBBB or LBBB for *possible* ischemia/infarction. In **Figure 05.25-1** — we reproduce the ST-T wave changes to look for:

- Normally with **LBBB/RBBB** — the **ST-T wave** will be ***oppositely directed*** to the ***last QRS deflection*** in the 3 **KEY** leads (*leads I, V1, V6*).
- **NOTE:** With **IVCD** — the *ST opposition rule* does not consistently work (*reliability for ischemia/infarction is more limited*).

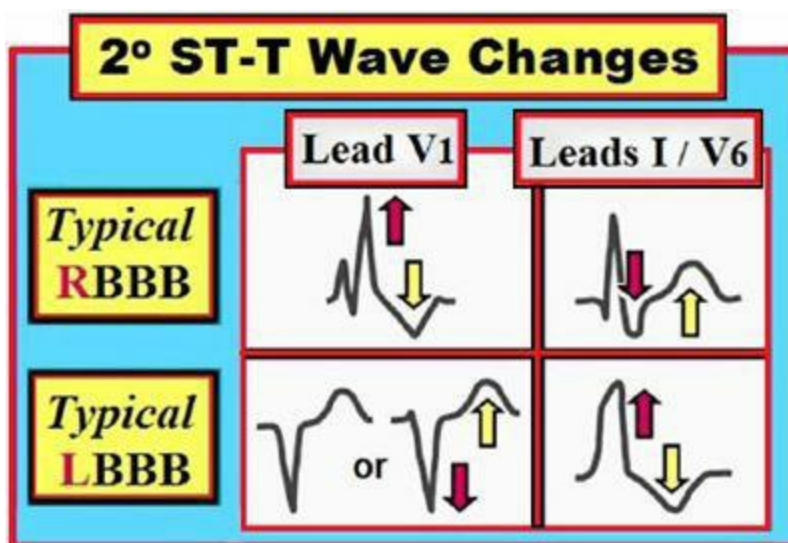


Figure 05.25-1: The *expected* ST-T wave response to either *typical* RBBB or *typical* LBBB is “***ST opposition***”. That is — the ST segment *and* T wave should be ***oppositely directed*** to the ***last QRS deflection*** (*red and yellow arrows*) in each of the 3 **KEY** leads (= *leads I, V1, V6*). Deviation from this pattern is abnormal — and indicates a ***primary*** (1°) **ST-T wave change** (*that suggests ischemia or infarction*).

05.26 – RBBB: You Can See Q Waves!

Clinically — it will usually be *easier* to diagnose ischemia/infarction when there is *underlying*

RBBB than when there is LBBB. This is because RBBB does not alter the *initial* direction of septal depolarization as LBBB does (Section 05.7).

- As the *initial* part of the QRS complex — **Q waves** are written at an *early* point during the process of ventricular activation. Because the conduction defect of **RBBB** does not alter the *left-to-right* direction of *normal* septal activation (*since the right bundle branch goes down the right side of the septum*) — the presence of RBBB will usually not prevent inscription of Q waves. This is in contrast to LBBB that does change the direction of *initial* septal activation (Section 05.28). **KEY Point:** Much (*most*) of the time — You can see infarction Q waves even when *complete* RBBB is present (Figure 05.26-1). You may or may not see acute changes.

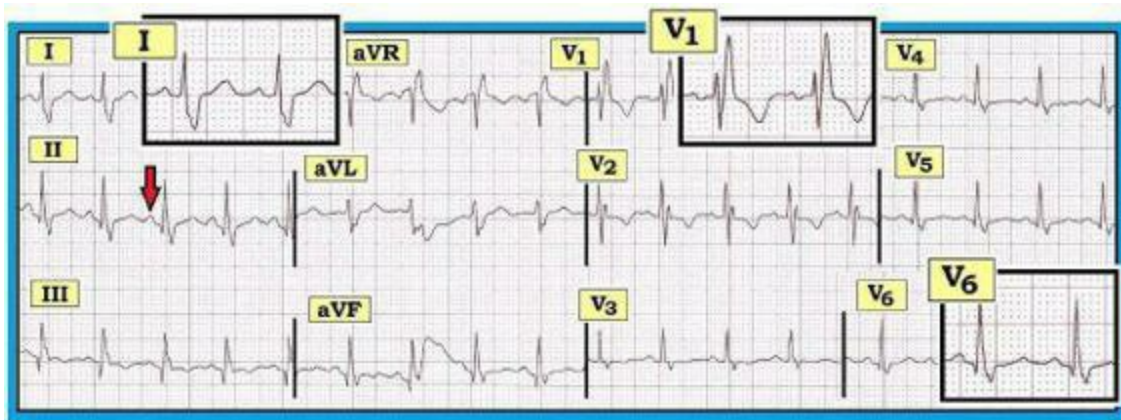


Figure 05.26-1: Sinus tachycardia with *complete* RBBB. Despite RBBB — **Q waves** are seen in leads II,III,aVF; and V5,V6. This suggests *inferolateral* infarction has occurred at *some* point in the past. ST-T wave appearance suggests that the infarct is *less* likely to be acute (See text).

05.27 – Underlying RBBB: How to Diagnose Acute MI?

As stated in Section 05.24 — Diagnosis of ischemia/infarction is *always* more challenging when there is *underlying* BBB. This is because the typical *secondary* ST-T wave changes that result from BBB (Figure 05.25-1) — may *mask* ST-T wave changes due to ischemia/infarction. That said — it is important to *carefully* scrutinize the ECG, because chronic (*and even acute*) changes may at times be seen.

- Realize that it will always be more difficult to diagnose ischemia/infarction with LBBB than with RBBB. But — sometimes you'll be surprised! (*as we illustrate in Section 05.29*).

Consider the case of *complete* RBBB seen in **Figure 05.27**:

- The rhythm in **Figure 05.27** is regular and the QRS complex is wide. Although we do *not* see upright P waves in lead II — the *mechanism* of this rhythm is clearly *supraventricular* — as evidenced by upright P waves with *fixed* PR interval that are seen to *regularly* occur in *simultaneously* recorded leads V1,V2,V3.

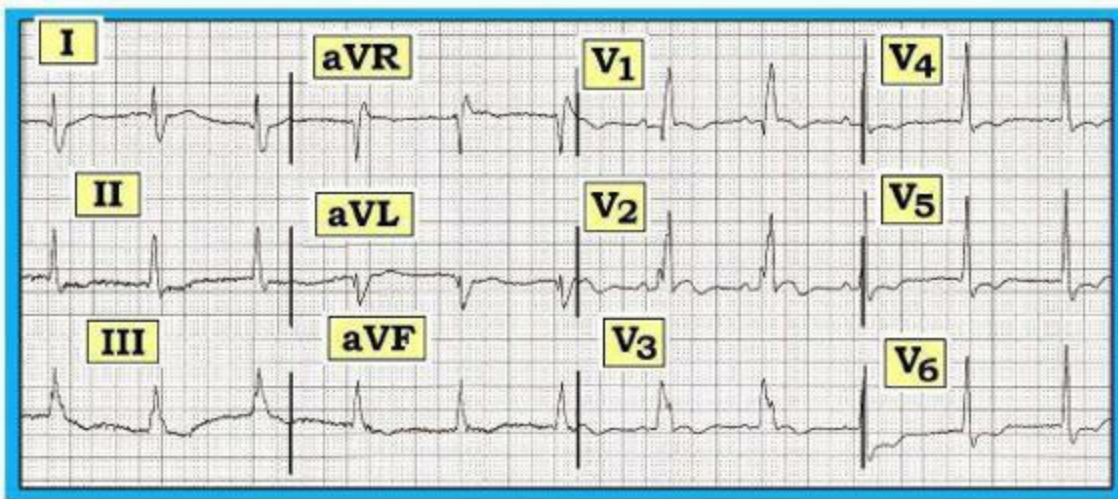


Figure 05.27-1: Supraventricular rhythm with **complete RBBB**. Despite RBBB — there is evidence of infarction that may be acute (*See text*).

Analysis of the ECG in Figure 05.27-1: Although *uncertain* about the precise mechanism of this rhythm — *unmistakable* P waves in *simultaneously* recorded leads V1,V2,V3 define the rhythm to be supraventricular. Perhaps artifact undulations in the baseline of lead II prevent sinus P waves from being seen or perhaps this is a *low atrial* rhythm? Regardless — We proceed by assessing the *reason* for QRS widening according to the **Algorithm** put forth at the beginning of this Section on BBB (Figure 05.2-1).

- A predominantly *upright* wide QR complex is seen in lead V1. This qualifies as an **RBBB “equivalent” pattern** (Section 05.16).
- The diagnosis of **complete RBBB** is secured by noting the presence of wide terminal S waves in *both* leads I and V6.
- **Q waves** — are seen in several leads of this tracing. The q wave in lead I is small, and of uncertain significance. However, the qrS pattern in lead aVL is distinctly abnormal and highly suggestive of lateral infarction. In addition — the **QR pattern** in **lead V1** in the setting of RBBB is virtually diagnostic of prior anterior (*or anteroseptal*) infarction.
- **ST-T waves** — are not normal for RBBB. Although the small amplitude upright T wave in lead I is consistent with the **ST opposition rule** (Fig. 05.25-1) — ST depression in **lead V6** and *subtle-but-real* ST elevation in **lead V1** are *both* contrary to what one normally expects with RBBB.
- By the concept of “*neighboring*” leads — We see similar ST depression with shallow T inversion extending from V6 to V4,V5 — and ST coving with slight elevation not only in V1, but also in V2,V3.
- **Bottom Line:** *Despite* RBBB — We are strongly suspicious of previous and perhaps recent (*if not acute ongoing*) infarction in Figure 05.27-1. History and comparison with *prior* tracings would be invaluable for clarifying what is likely to be new vs old.

05.28 – Underlying LBBB: How to Diagnose Acute MI?

Special considerations are needed to assess for ischemia/infarction when there is **LBBB**. These are highlighted in the *schematic* tracings shown in Figure 05.28-1.

- Diagnosis of **LBBB** is confirmed in **Panel A** of **Fig. 05.28-1** — by the finding sinus rhythm with QRS widening showing a *negative* QRS complex in lead V1 and a monophasic *upright* R wave in leads I,V6 (*Section 05.6*).
- Note in this *schematic* example of **typical LBBB** (**Panel A**) — that there are QS complexes and ST elevation in leads V1,V2,V3 that simulate *anterior* infarction — and that ST-T wave depression in *lateral* leads simulates ischemia. All of these findings are commonly seen with LBBB.
- **Key Point:** There is *no* ECG evidence of ischemia/infarction in **Panel A** of **Figure 05.28-1**. Instead — All one can say is that there is LBBB.

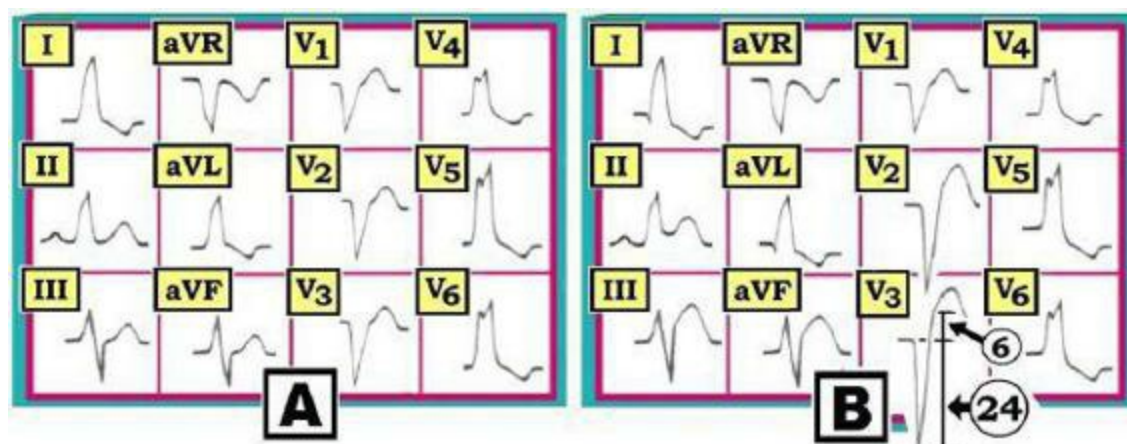


Figure 05.28-1: *Schematic* illustration of **typical LBBB** (**Panel A**) — and of LBBB with ECG signs of **acute STEMI** (**Panel B**).

Clinical Considerations: The main thing one cares about in assessing a patient with **LBBB** who presents to the ED (*Emergency Department*) with **new-onset chest pain** — is whether there is acute coronary occlusion (**ST Elevation Myocardial Infarction-equivalent**) necessitating immediate intervention (*cath lab activation; angioplasty/stenting*).

- Overall — *less* than 5% of patients with LBBB and *new-onset* chest pain have acute coronary occlusion. Although insensitive — the finding of **primary ST elevation** is highly *specific* for acute coronary occlusion. This is seen in leads II,III,aVF of **Panel B** in **Figure 05.28-1**.
- Further support of likely acute coronary occlusion may be forthcoming — IF there is **excessive discordance**. That is — IF in leads V1,V2, or V3 you can see a clear J point where the QRS ends and the ST segment begins — and — IF the J point of the ST segment is up or down >20% of QRS amplitude — then there is *excessive discordance* (*by the Smith-modified Sgarbossa criterion*). This criterion is satisfied in **lead V3** of **Panel B** (*J-point elevation* = 6; *S wave depth* = 24 — and $6/24 > 20\%$). **CAVEAT** — it may be difficult to know where the J-point is if anterior ST elevation with LBBB is of *smooth* contour (*in which case you won't be able to use this criterion*).

NOTE: Debate continues as to whether the ECG finding of **LBBB** of **uncertain age** in a patient with *new-onset* chest pain is sufficient to justify acute cath lab activation.

- Most such patients do *not* have acute coronary occlusion. Some may have troponin elevation (*that satisfies the definition of acute MI*) — but overall, initial management of patients *without* acute ECG changes will be *similar* to that of patients admitted to the hospital with a “*rule out MI*” diagnosis.
- Awareness of the need to look for *primary* ST elevation and *excessive* discordance in chest pain patients with LBBB will hopefully help to identify the important minority likely to have *acute* coronary occlusion.

Analysis of Other Leads in Panel B: In addition to the *excessive* discordance described above — there are findings in *other* leads in **Panel B** that *strongly* suggest *acute* ischemia/infarction (**Figure 05.28-2**):

- *How many of these other findings can you identify?*

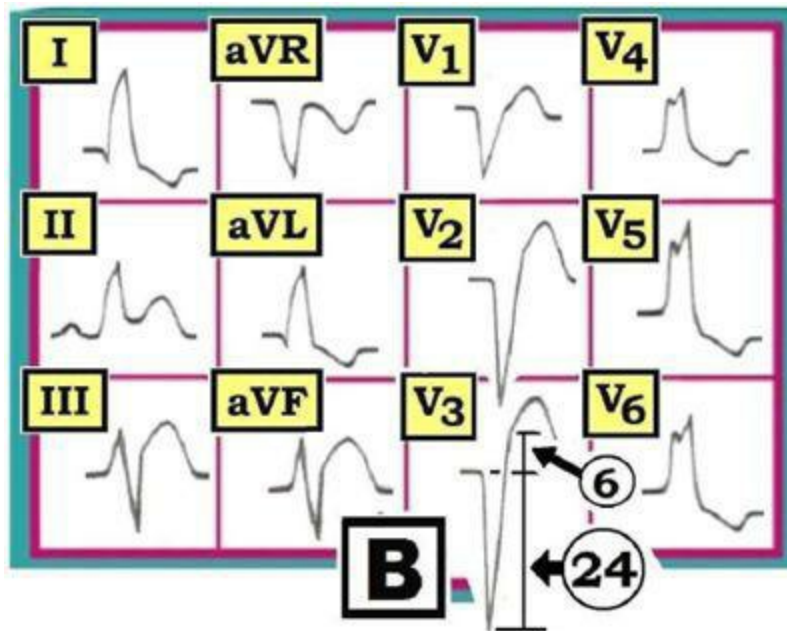


Figure 05.28-2: In addition to *excessive* discordance for the ST segment in lead V3 — What *other* findings strongly suggest *acute* ischemia/infarction?

ANSWER to Panel B in Figure 05.28-2: Assessment of the *schematic* tracing in Panel B summarizes essential points covered thus far:

- QRS morphology in the 3 key leads is consistent with **LBBB** (*upright QRS in leads I,V6; negative QRS in lead V1*).
- Look next at **ST opposition** in the 3 key leads. ST-T waves are *oppositely* directed to the last QRS deflection (*as they should be*) in leads I,V1,V6.
- There should *never* normally be *lateral* q waves with LBBB (*Section 05.6*). Finding a **Q wave** in lead **I**, **aVL**, **V5**, or **V6** means an infarct has *at some point* occurred. Note the Q wave in leads I and aVL in **Panel B**.
- Although *not* common — **primary ST elevation** is the most reliable indicator of *acute* STEMI with LBBB. This is seen in leads II,III,aVF of **Panel Panel B**. Therefore, *despite* LBBB — the

*schematic ECG seen in Figure 05.28-2 (= Panel B) is **diagnostic** of **acute inferior STEMI**.*

- In addition to *excessive* discordance for the amount of *anterior* ST elevation — there may also be *excessive* discordance for the amount of *lateral* ST depression. That said — *primary* ST elevation in the *inferior* leads of this tracing is far more reliable as an indicator of *acute STEMI* with LBBB.

05.29 – FIGURE 05.29-1: *Acute STEMI despite LBBB/RBBB?*

We conclude this section on assessing patients with RBBB and LBBB for ECG evidence of *acute* ischemia/infarction — with a series of 4 ECGs, beginning with the 2 tracings shown in **Figure 05.29-1**.

- *Both* of the ECGs in Figure 05.29-1 were obtained from patients with *new-onset* chest pain and *known* LBBB. Despite underlying LBBB — Is there ECG evidence of *acute STEMI* on either tracing? *Should the cath lab be activated?*

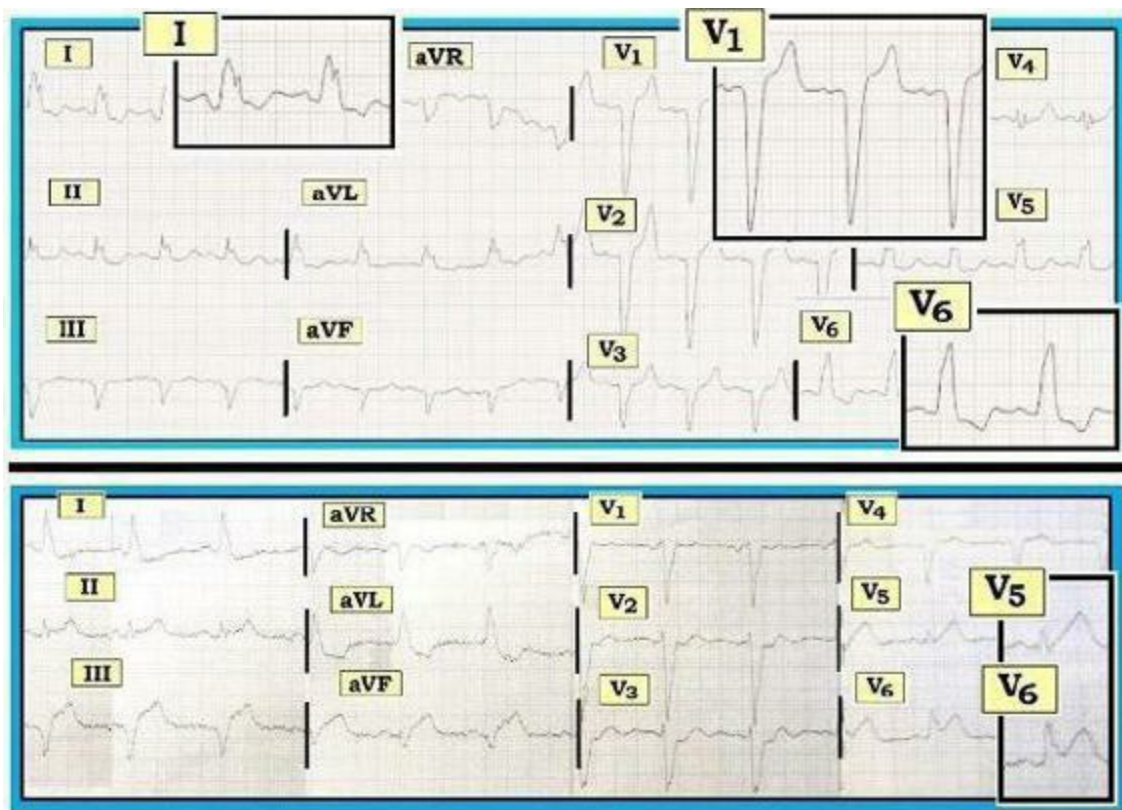


Figure 05.29-1: ECGs obtained from 2 patients with *baseline* LBBB and *new-onset* chest pain. Is there evidence of *acute* MI in *either* tracing?

ANSWER to Figure 05.29-1: Sinus rhythm with LBBB is present in *both* ECGs shown above. Despite Q waves in leads III,aVF; and in V1,V2,V3 — there is *no evidence* of acute STEMI in the **Top ECG**. Instead, ST-T waves are appropriately *opposite* (Figure 05.25-1) — and there is no excessive discordance. The Q waves that are seen in leads V1,V2,V3 are expected with LBBB, and those that are seen in leads III and aVF are *not* necessarily abnormal given the presence of this conduction defect.

- **Bottom ECG** — shows **LBBB** with **acute STEMI**! Note *primary* ST elevation in leads II,III,aVF; and especially in leads V5,V6. *Activate the cath lab!*

Now consider the example of **RBBB** shown in **Figure 05.29-2**. This tracing was obtained from a patient with *new-onset* chest pain. In addition to complete RBBB — Is there *also* evidence of an *acute* STEMI?

- *How certain* are you of your diagnosis?

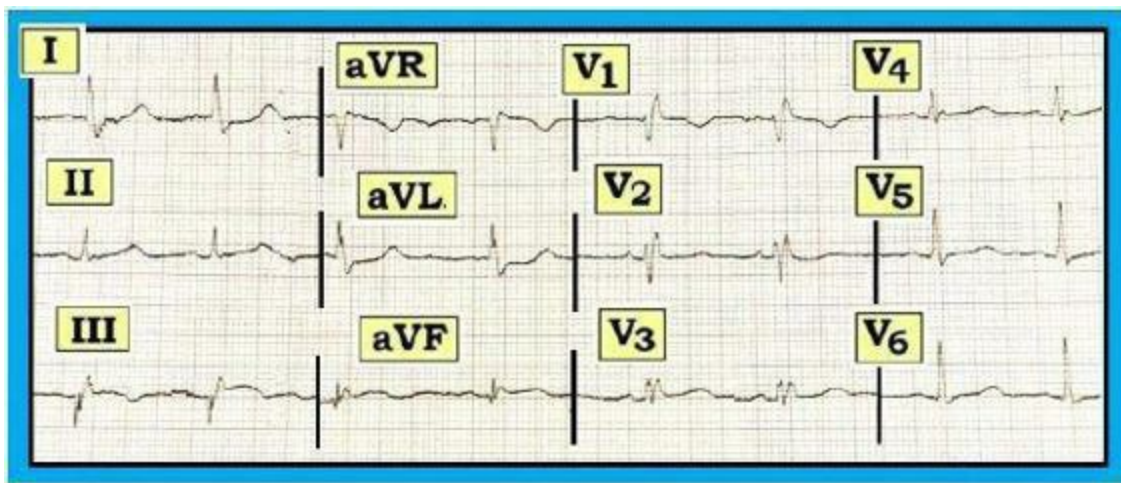


Figure 05.29-2: ECG obtained from a patient with *new-onset* chest pain. In addition to *complete* RBBB — Is there *also* evidence of an *acute* STEMI? (See text).

ANSWER to Figure 05.29-2: The rhythm is sinus arrhythmia. Complete RBBB is diagnosed by QRS widening with an rSR' in V1 plus wide terminal S waves in leads I,V6.

- There is *subtle-but-real* ST coving and elevation in lead V1 *that should not be*. Support that this finding is real — is forthcoming from *slight-but-definite* ST segment coving and elevation in lead III (*and to a lesser extent in aVF*) — plus *reciprocal* ST depression in lead aVL.
- A deep Q wave is already present in lead III.
- We interpret this tracing as sinus arrhythmia; **RBBB**; and **probable acute ongoing inferior STEMI**.
- Beyond-the-Core: We suspect acute *proximal* RCA (*Right Coronary Artery*) occlusion, possibly with acute right ventricular MI (*given subtle ST elevation in lead V1 in the setting of acute inferior infarction*).

Finally, consider the conduction defect shown in **Figure 05.29-3** — obtained from a patient with chest pain. Despite LBBB/IVCD — Is there evidence of an *acute* STEMI?

- *How certain* are you of your diagnosis?

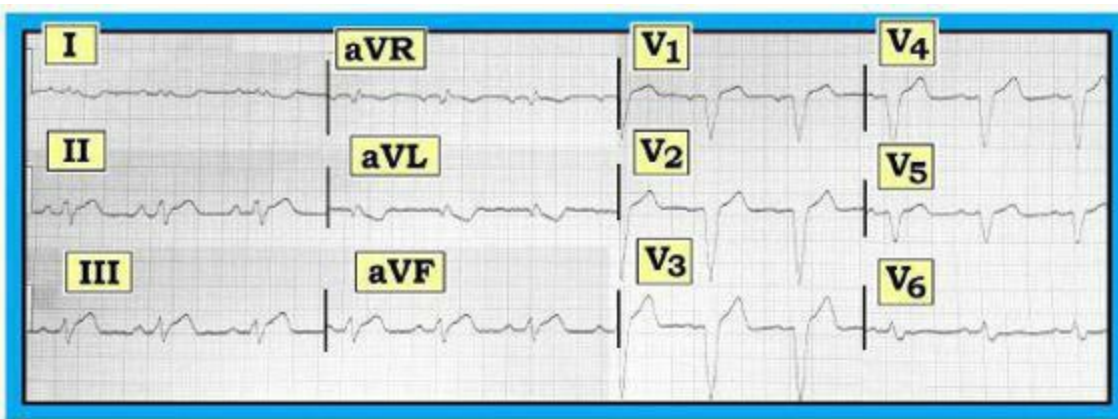


Figure 05.29-3: ECG obtained from a patient with *new-onset* chest pain. In addition to the conduction defect — Is there *also* evidence of an *acute* STEMI? (My appreciation to Ben Beuchler for allowing me to publish this ECG)

ANSWER to Figure 05.29-3: The rhythm is sinus at ~75/minute. The QRS complex is wide. Although the *anterior* precordial leads look like LBBB (*deep QS complexes*) — R wave amplitude in lead I is tiny, and there is a terminal S wave in V6. Our preference would be to call the conduction defect = “**IVCD**” (*IntraVentricular Conduction Defect*) — though we would accept *either* IVCD *or* LBBB as correct interpretations. In either case — the anterior QS complexes seen are *not* diagnostic of prior infarction given the conduction defect.

- Regardless of the terminology used (*LBBB or IVCD*) — the important clinical point is that interpretation of acute ischemia/infarction is more difficult in the setting of this type of conduction defect. Thus, the ST segment elevation seen in leads V1-through-V4 of Figure 05.29-3 is *not* a reliable predictor of acute anterior infarction, since LBBB *commonly* manifests anterior ST elevation comparable in shape and amount to that seen here.
- On the other hand — there should *not* be **ST elevation** in the *inferior* leads (**Figure 05.29-4**). Note J-point ST elevation is present in *each* of the inferior leads (*red arrows in blow-up inserts of Fig. 05.29-4*). In support that this inferior ST elevation is real — is *mirror-image* shape **reciprocal ST depression** in lead aVL (*red circle*). In addition — it looks like a Q wave has already formed in lead aVL. Therefore — this ECG strongly suggests *acute* inferior STEMI *despite* the presence of *underlying* LBBB/IVCD.

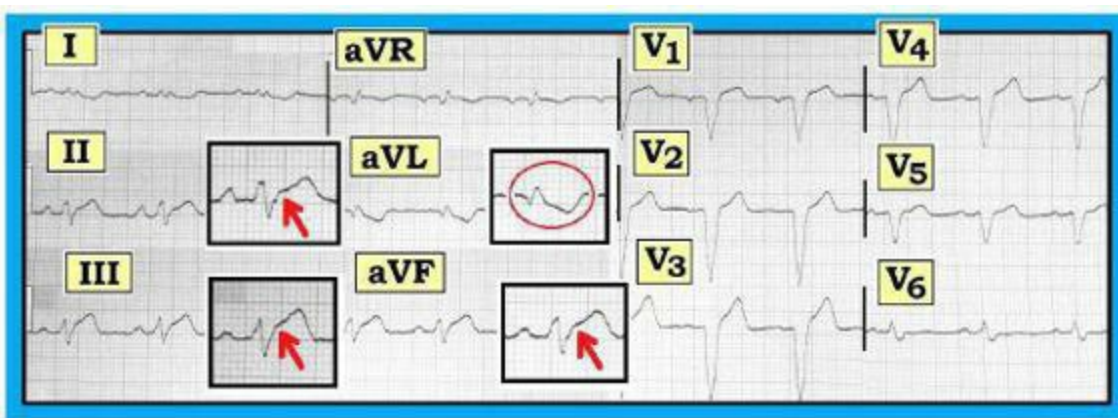


Figure 05.29-4: Addition of *blow-up* inserts to Figure 05.29-3, from this patient with chest pain. We would accept *either* IVCD *or* LBBB as explanations for the conduction defect. While no conclusions

can be drawn from the ST elevation seen in anterior precordial leads — ***acute STEMI*** can be diagnosed from the ST elevation seen in *each* of the inferior leads (*red arrows*) with a Q wave and mirror-image *reciprocal* ST-T wave changes in lead aVL (*See text*).



ECG Diagnosis of ventricular chamber enlargement becomes *difficult* in the presence of **conduction defects** (*RBBB*, *LBBB*, *IVCD*). Criteria for LVH/RVH are based on the *normal* sequence and progression of ventricular activation. All of this *changes* in the presence of bundle branch block.

- Instead of *near-simultaneous* left and right ventricular activation — there will be *delay* in activation of that part (*or those parts*) of the ventricles served by the nonfunctioning conduction fascicle(s). This alteration in time sequence of the *relative* contribution of left and right ventricular forces *invalidates* numeric and morphologic criteria for chamber enlargement that were derived during sinus rhythm with *intact* ventricular conduction.

BOTTOM Line: Traditional ECG criteria for assessment of *ventricular* chamber enlargement should *not* be used when there is BBB/IVCD. One *never* quite knows how much of the increase in QRS amplitude seen is a result of ventricular hypertrophy vs scarring, prior infarction *and/or* alteration in sequence (*and therefore relative contribution*) of ventricular depolarization forces.

- On the other hand — **criteria** for LAA/RAA are *unchanged* by BBB/IVCD (*Sections 08.14-through-08.22*).
- Clinically — IF you really need to know about atrial or ventricular chamber dimensions — *Get an Echocardiogram!*

NOTE: What follows in Sections 05.31 and 05.32 constitutes *advanced* material that may be **Beyond-the-Core** for the usual provider. We accept a preference by some to *simplify* ECG interpretation and *skip* the step of assessing ventricular enlargement when there is BBB because of the difficulty validating ECG criteria in the presence of conduction defects.

- That said, for those with an interest in going *Beyond-the-Core* — *Read on!* There are ways with *high* reliability to diagnose *probable* LVH despite the presence of LBBB or RBBB.

05.31 – LBBB: What Criteria to Use for LVH/RVH?

Most patients with **LBBB** have *underlying* heart disease. IF there is *longterm* hypertension, cardiomyopathy or heart failure and LBBB — the prevalence of **LVH** is **~80%**, even before one looks at the ECG.

- The probability that a patient with LBBB has LVH goes up to **>90%** — IF **with LBBB** there are **very deep S waves** ($\geq 25\text{-}30\text{mm}$) in **V1, V2 or V3** (Figure 05.31-1).
- The likelihood of LBBB plus LVH also increases IF the patient has **LAA** (*Left Atrial Abnormality*) — as evidenced by a deep *negative* component to the P wave in lead V1.

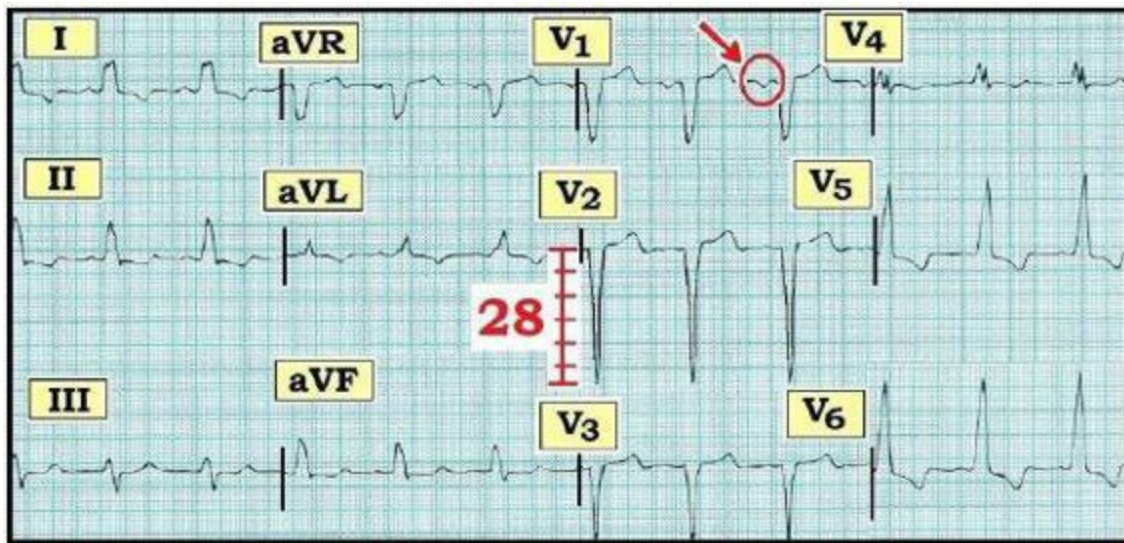


Figure 05.31-1: Sinus rhythm with **LBBB** plus *probable* LVH because: i) the deep *negative* component to the P wave in lead V1 suggests **LAA** (circle) and ii) the **S wave** in lead V2 is *very deep* (>25mm). **NOTE:** *Either* LAA *or* the deep S wave in V1,V2,V3 alone would have been enough to increase likelihood of LVH.

Semantic Point: We favor use of the terminology, “*probable*” LVH — for the ECG picture seen in Figure 05.31-1, in which there is LBBB *plus* a *very* deep S wave in lead V2. Although statistical likelihood that this patient has LVH is *at least* 90% (*actually more since there is also LAA*) — the ECG diagnosis of “LVH” is based more on *prevalence* than on specific ECG voltage criteria. Use of the modifier “*probable*” acknowledges the uncertainties of diagnosing LVH in the presence of this conduction defect.

- **How Deep the S Wave?** Note that we *cannot* tell from Figure 05.31-1 how deep the S wave in lead V3 would be — since the *bottom* of the S wave in this lead has been cut off.

Clinical Notes: Consider the following additional points:

- The presence of **LBBB** may mask and mimic other conditions. We’ve already discussed *special* considerations for assessing ischemia/infarction with LBBB (Section 05.28). For example — *despite* the QS complex in leads V1,V2,V3 of Figure 05.31-1 — We can *not* diagnose prior *anterior* infarction. This is because the patient has **complete LBBB**. Anterior precordial QS complexes with poor R wave progression are an *expected* accompaniment of *complete* LBBB (Section 05.8).
- In addition — We **can not comment** on the possibility of LV “**strain**” in the presence of LBBB. As highlighted by the ST Opposition Rule (Section 05.25) — *lateral* ST-T wave depression is an *expected* finding in LBBB. Its presence or absence says nothing about whether or not there is associated LV “strain”. Thus, even though we would interpret the ECG in Figure 05.31-1 as suggestive of *probable* LVH (*because of the deep S in V2 plus LAA*) — the ST-T depression seen in leads I,aVL,V5,V6 of this tracing is of *no* support to our interpretation. All we can say is, “LBBB with probable LVH”.

- Finally — Realize that it is virtually *impossible* to *diagnose* RVH when there is LBBB. The LBBB simply masks right ventricular events.

05.32 – RBBB: What Criteria to Use for LVH/RVH?

ECG diagnosis of ventricular chamber enlargement is equally challenging when there is **RBBB**. Once again — difficulty arises because the *altered* sequence of ventricular activation leaves us *uncertain* as to how much any increase in QRS amplitude might be due to ventricular hypertrophy *vs* delay (*and therefore nonopposition*) of right ventricular depolarization forces. Realizing the imperfection inherent with *any* proposed ECG criteria for chamber enlargement when there is BBB — We make the following points:

- **Complete RBBB** is a *terminal delay* (Sections 05.4; 05.5). As such — it does *not* affect the initial parts of ventricular activation (*during which time the septum and then left ventricle are activated*). As a result — the process of LV activation is virtually intact when there is RBBB with the exception that the larger left ventricle will *no longer* be opposed by smaller RV forces that are delayed. **Bottom Line:** ECG voltage criteria for LVH in the presence of *complete* RBBB are probably *not* that different in certain leads compared to voltage criteria when there is no conduction defect.
- **Suspect LVH** (*despite* RBBB) — IF the R wave in lead **aVL** is ≥ 12 , or the R wave in lead **V5** or **V6** is ≥ 25 .
- Depth of the S wave in leads V1,V2 is *no longer* of assistance for assessing LVH when there is RBBB — because the rSR' in lead V1 essentially *precludes* S wave formation.

Consider the ECG in **Figure 05.32-1**. There is a low atrial rhythm with QRS widening consistent with **complete RBBB** (rSR' in V1; wide terminal S waves in leads I,V6).

- Is there ECG evidence of ventricular enlargement in Figure 05.32-1?

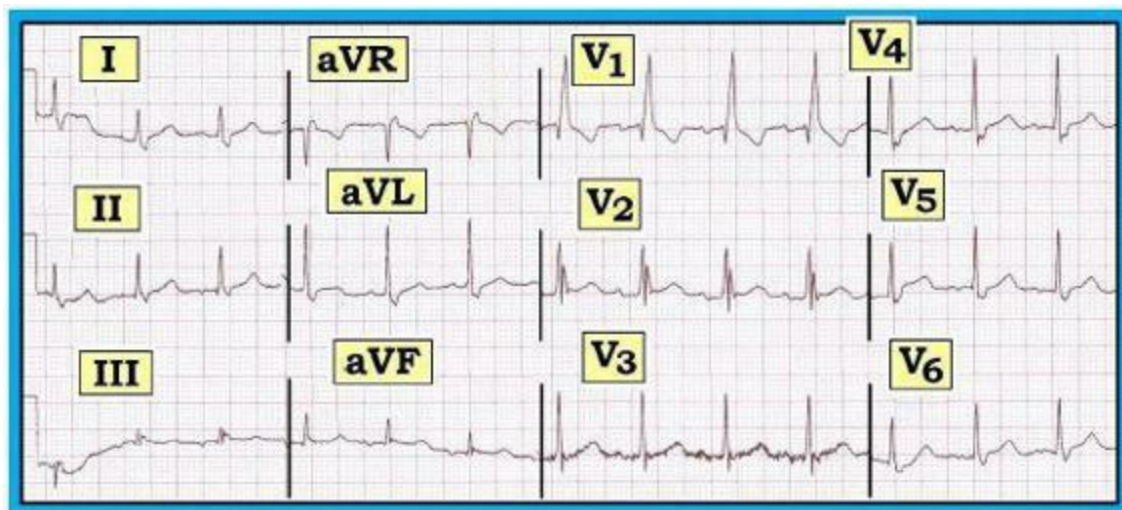


Figure 05.32-1: Low atrial rhythm with **complete RBBB**. Is there ECG evidence of ventricular

enlargement? (*See text*).

Answer to Figure 05.32-1: ECG criteria for the diagnosis of LVH are covered in detail in Sections 08.1-through-08.13. S wave depth typically increases in leads V1,V2 with LVH — reflecting an increase in LV forces moving *away* from these *right-sided* leads. That said — the presence of *complete* RBBB in Figure 05.32-1 *negates* formation of S waves in leads V1,V2. *Other* leads must therefore be used to assess the possibility of LVH.

- R wave amplitude is *not* increased in leads V5,V6 of Figure 05.32-1.
- On the other hand — the R wave in lead aVL *does* attain *at least* 12mm in height, and therefore satisfies criteria for ***probable* LVH** in this patient with RBBB.
- NOTE: We qualify our assessment in Figure 05.32-1 with the term, “***probable***” LVH — to acknowledge *reduced* specificity of voltage criteria for LVH in the presence of *complete* RBBB. We simply don’t know how much of the increase in R wave amplitude in aVL is due to anatomic LV enlargement *vs* *nonopposition* of LV depolarization forces due to the conduction defect.

Is there RVH in Figure 05.32-1? By definition — ECG diagnosis of RVH in the presence of RBBB is problematic. This is because the presence of this conduction defect totally *changes* the sequence and nature of RV depolarization. RV depolarization with RBBB is *delayed* and *unopposed* by LV depolarization that will usually be complete by the time the activation wavefront finally makes its way into the right ventricle. As a result — the tiny right ventricle may sometimes generate a surprisingly *tall* R’ deflection in lead V1 (*because there no longer is opposition from LV forces*). **KEY Point:** The height of the R’ deflection in lead V1 with complete RBBB is not necessarily related to the size/thickness of the right ventricle.

- Realize that *some* electrocardiographers cite R’ amplitude of >10mm (*or* >15mm) in lead V1 as a criterion for diagnosing RVH in the presence of RBBB. We are not in favor of this practice — because it will result in false positive diagnosis of RVH in many cases.
- **Our Preference** — is to ***undercall* RVH** in the presence of **RBBB**. We generally do *not* indicate “*probable RVH*” in the setting of RBBB *unless* in addition to a very tall R’ component in lead V1 (*of* >10-15mm) — there is *also* RAA (*Right Atrial Abnormality*) in a patient *known* to have longstanding pulmonary disease. **BOTTOM Line:** While we acknowledge that some interpreters may say RVH is present in Figure 05.32-1 because of the tall R’ component in lead V1 — We would not do so because: **i)** *No* history of pulmonary disease is given; and **ii)** there is *no* sign of RAA.

05.33 – Brugada Syndrome



Brugada Syndrome

First described in 1992 — the **Brugada Syndrome** is important to recognize because of an associated very **high risk** of **sudden death** in otherwise healthy young or middle-aged adults who have structurally normal hearts.

- The prevalence of Brugada Syndrome in the general population is $\sim 1/2,000$. The syndrome has become a leading cause of sudden death in young adults (*under 40 years of age*).
- Given heightened awareness and recent *increased* attention directed toward early recognition — We suspect the estimated prevalence for Brugada syndrome will continue to increase in the years-to-come.

05.34 – ECG Recognition: *Distinction Between Type I and II*

Distinction is made between 2 types of Brugada ECG patterns:

- **Type I** (seen in **Panel A** of Figure 05.34-1) — is **diagnostic** of Brugada Syndrome — because it shows $\geq 2\text{mm}$ coved ST elevation with *sharp* downslope plus T wave inversion in ≥ 2 anterior leads. *This is not RBBB.*
- **Type II** (in **Panel B**) — is **not diagnostic** of Brugada Syndrome — unless the *saddleback* anterior ST elevation with *positive* T wave (seen here in leads V2, V3) at some time later *converts* to a Type I tracing. Most often — this *concave up* ST elevation is due to **benign early repolarization** (assuming no syncope, arrhythmia or familial history of sudden death at an early age).

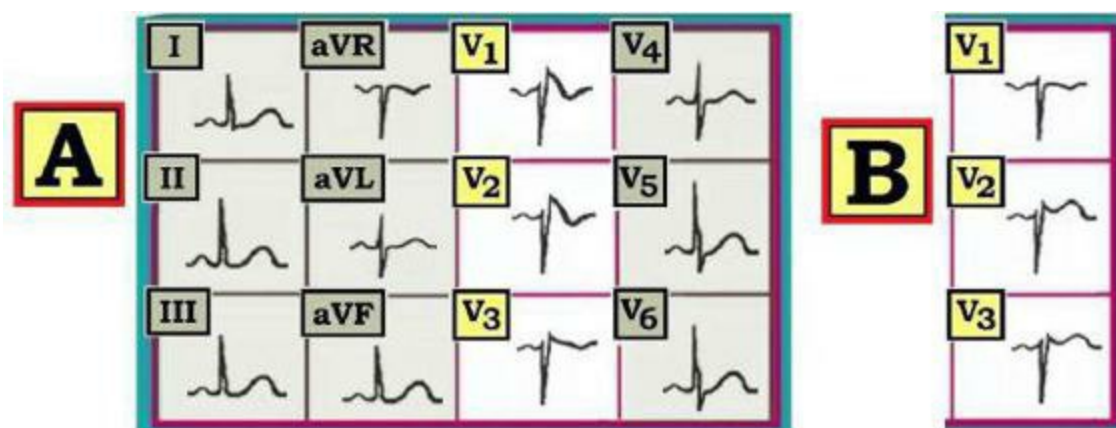


Figure 05.34-1: Leads V1, V2 in **Panel A** are *diagnostic* of Brugada Syndrome. The *saddleback* pattern in leads V2, V3 of **Panel B** is not.

CAVEAT: Type I or Type II Brugada ECG patterns may be induced by a variety of factors. A partial list includes the following:

- *Certain drugs (antiarrhythmics; calcium channel blockers; β -blockers; antianginals; psychotropic medications; alcohol; cocaine; other drugs).*
- Acute febrile illness.
- Variations in autonomic tone.
- Hypothermia.
- Electrolyte imbalance (*hypokalemia*).
- Ischemia/infarction.
- Cardioversion/defibrillation.
- Bradycardia.
- *Repositioning precordial leads (recording leads V1, V2 from the 2nd or 3rd intercostal space).*

NOTE: Whether clinical implications of a transient *induced* Brugada pattern (*Type I or Type II ECG*) are the same as with its spontaneous occurrence is uncertain — and probably depends on *associated* clinical parameters. Thus, *transient* induction of a Type II ECG pattern would seem *less* likely to be important if only briefly seen during acute febrile illness in an otherwise healthy young adult with negative family history. In contrast — induction of *longer-lasting* Type I ECG changes in an older patient with syncope and positive family history is unquestionably of great concern. Definitive answers regarding clinical implications have not yet been worked out for all situations — so judgment is needed in clinical decision-making.

05.35 – *WHAT to DO? - when a Brugada Pattern is Found?*

The clinical question that arises is the following: “*What to do?*” — IF you encounter a patient with a Type I or Type II tracing?

- Patients found to have a **Type I ECG** (*even if asymptomatic*) — should probably be referred to a cardiologist. **EP** (*ElectroPhysiologic*) **study** is clearly indicated when there are symptoms (*seizure; syncope or presyncope; arrhythmia*) or for positive family history of sudden death at an early age (*below 50*). An **ICD** (*Implantable Cardioverter Defibrillator*) is indicated IF the patient has *inducible* VT (**Figure 05.35-1**).
- Follow-up with a *knowledgeable* primary care clinician (*but without necessarily referring to cardiology*) may be appropriate — IF a *non-diagnostic* pattern is seen in an otherwise healthy adult with normal exam and *negative* family history (**Figure 05.35-2**).
- ***IF in doubt*** about *What to Do?* for a patient you encounter who has a *Brugada-type* ECG — it is prudent to err on the side of caution. This entails consulting a clinician *experienced* in diagnosis and management of Brugada syndrome, its variants and its mimics.

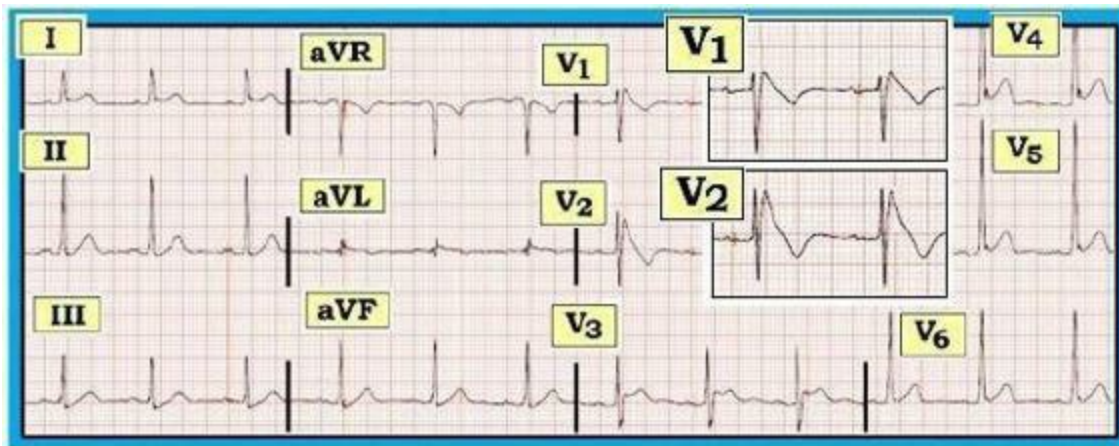


Figure 05.35-1: ECG obtained from a young adult with syncope. Even without a positive family history for sudden death at an early age — the **Type I Brugada pattern** seen here in leads V1,V2 is enough to justify *prompt* referral to an EP (*ElectroPhysiology*) cardiologist for consideration of ICD placement. Note J-point ST elevation of 2-3mm in lead V1 and more than 5mm in V2 — followed by convincingly *steep* ST downslope into *inverted* T waves (*comparable to Panel A in Fig. 05.34-1*). This ECG is *diagnostic* for **Brugada Syndrome**.

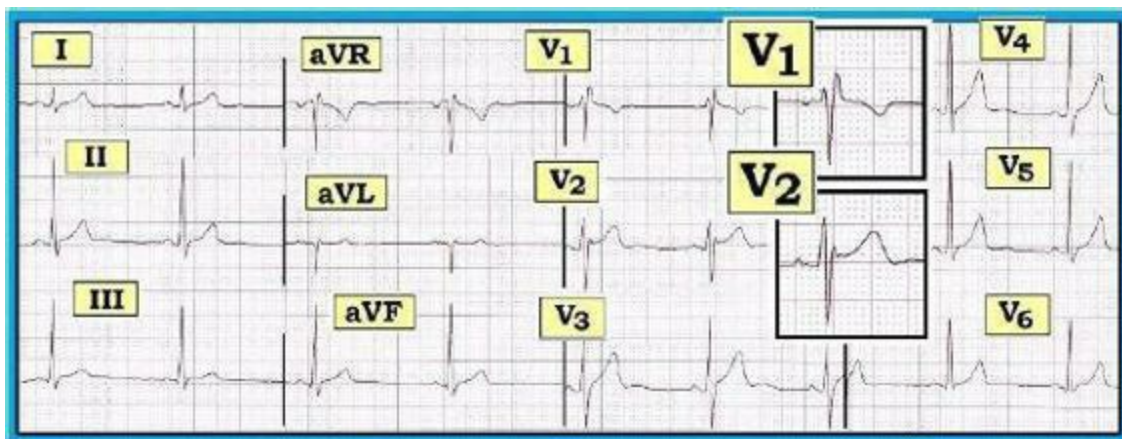


Figure 05.35-2: ECG obtained from an *asymptomatic* young adult. An rSr' pattern is seen in lead V1 with *saddleback* ST elevation in lead V2. This ECG is consistent with a **Type II Brugada pattern**. Although more than is usually seen with simple *incomplete* RBBB or early repolarization — the ECG pattern in this figure is ***not* diagnostic** of Brugada syndrome. Referral and ICD placement are *not* necessarily needed — especially if this asymptomatic young adult has a *negative* family history. **Bottom Line:** Judgment and clinical correlation are needed for optimal evaluation and management. *Not all answers are yet in.* Each case should be individualized (*See text*).

05.36 – WPW (Wolff-Parkinson-White)



WPW (Wolff-Parkinson-White)

In the setting of normal sinus rhythm — the only exception to the *simplified Algorithm* for assessment of **QRS Widening** (Section 05.2) — is the Wolff-Parkinson-White (WPW) Syndrome.

- Although admittedly uncommon (~2 per 1,000 in the general population) — WPW occurs just often enough to cause problems for the unwary. The **importance** of WPW is twofold: i) It is “*the great mimic*” — and may simulate other conditions (*such as ischemia/infarction, hypertrophy and/or conduction defects*) — IF it is *not* recognized; and ii) The presence of one or more accessory pathways **predisposes** the patient to a number of potentially important **cardiac arrhythmias**.

05.37 – WPW: Pathophysiology / ECG Recognition

WPW — is a syndrome in which one or more *accessory* pathways exist that allow an *alternate* route for transmission of the electrical impulse from atria to ventricles. Thus, with WPW — sinus impulses **bypass** the AV node via an AP (Accessory Pathway) — and therefore arrive *early* in the ventricles (Panel A in Figure 05.37-1).

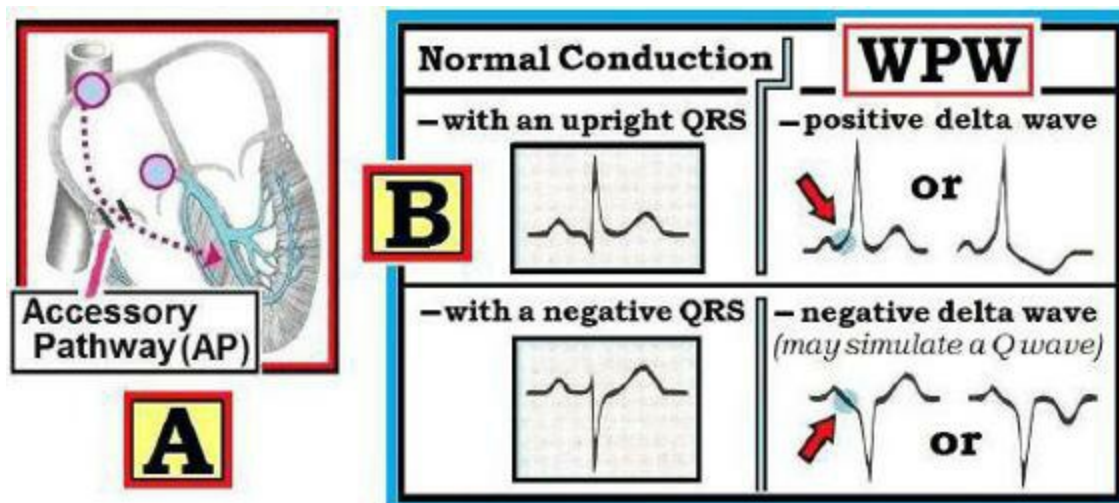


Figure 05.37-1: With WPW — impulses *bypass* the AV node via AP conduction. Although the AP shown in Panel A is *right-sided* — it could be left-sided, anterior or posterior and at times there is *more* than a single AP. The **3 ECG signs** of WPW are seen in Panel B — i) a **delta wave** (*which may be positive or negative*); ii) **QRS widening**; and iii) a **short PR interval** (See text).

ECG Recognition of WPW: It is usually easy to recognize WPW on a baseline 12-lead ECG when conduction *completely* utilizes the AP (Accessory Pathway). There are **3 ECG features** to look for (Panel B in Figure 05.37-1):

- *Delta waves*.
- A *short PR interval*.

- QRS widening.

Delta Wave: The ***delta wave*** is recognized as a distortion of the *initial* portion of the QRS complex. It is due to the fact that the electrical impulse *bypasses* the AV node — and arrives at the ventricles *directly* via conduction over the accessory pathway.

- Delta waves may be upright (*positive*) or downward (*negative*) — depending on where in the heart the AP is located. When delta waves are negative — they may simulate the Q wave of myocardial infarction (Panel B in Fig. 05.37-1):
- Even when conduction is entirely over the AP — delta waves will *not* always be seen in every lead. Moreover, delta waves may come and go — since conduction over the AP may be intermittent. At times — conduction may *simultaneously* occur over both the normal and accessory pathway. When this happens — the ECG characteristics of WPW may be subtle because the contribution from conduction over the normal AV nodal pathway may predominate (*and thereby mask*) ECG features of pre-excitation.

Short PR Interval: The reason the **PR interval** is ***short*** with WPW — is that the AV node is bypassed. With normal conduction in sinus rhythm — the electrical impulse *slows down* as it passes through the AV node on its way to the ventricles. As a result — most of the PR interval normally consists of the time it takes for the impulse to traverse the AV node. The electrical impulse arrives at the ventricles *sooner* with WPW because the usual relative *delay* that occurs when passing through the AV node is *avoided* by conduction over the AP.

QRS Widening: The **QRS widens** with WPW — because *after* the impulse arrives at the ventricles (*via conduction over the AP*) — it must travel over *nonspecialized* myocardial tissue until such time that it attains whatever distal portion of the conduction system that has *not* yet depolarized. Thus the delta wave may extend for 0.04 second or more (*reflecting slow conduction over nonspecialized myocardial tissue*). When the delta wave deflection is added to the rest of the QRS complex — the result is a *widened* complex.

Bottom Line Regarding ECG Recognition: Many variations exist on the above theme. Remember the following:

- WPW is *not* common in the general population — but it does occur (*and you will see it*)!
- When a patient with WPW is conducting over their accessory pathway — you can ***diagnose*** WPW by ***recognition*** of the following **3 ECG features** in at least *several* of the 12 leads of an ECG: **i)** QRS widening; **ii)** a *delta wave*; and **iii)** a *short PR interval*.
- **Preexcitation** (ie, *WPW conduction over an AP*) can be intermittent. There may be no indication on ECG that a patient has WPW if conduction is entirely (*or almost entirely*) over the normal AV nodal pathway at the time the tracing is recorded.

05.38 – WPW: The “Great Mimic” of other Conditions

As already emphasized — an essential reason for recognizing WPW is so that it is not confused with *other* conditions. This point is well illustrated by the *schematic* tracing shown in **Figure 05.38-1**:

- Note in Fig. 05.38-1 — that *delta* waves are not always present in all leads. For example, they are completely *missing* in leads I, aVL and V4, V5 of Figure 05.38-1.
- *Negative* delta waves are present in leads II, III, aVF and *simulate* inferior infarction.
- The tall R wave in lead V1 *simulates* RBBB.
- The tall, monophasic R wave in lead V6 *simulates* LBBB.
- ST-T wave depression in lead V6 *simulates* ischemia.

BOTTOM Line: It is *easy* to *overlook* WPW if one does not *routinely* use a *Systematic Approach*. This is especially true IF — conduction goes down *both* the AP and the normal AV nodal pathway at the *same* time (*in which case the delta wave may be subtle and the QRS may be no more than minimally widened*).

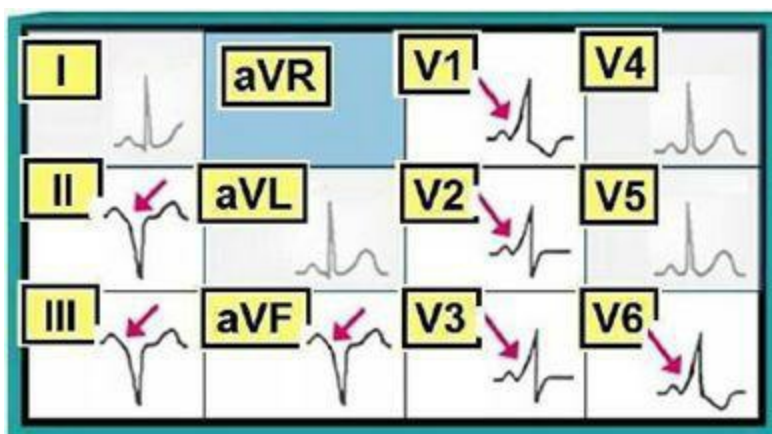


Figure 05.38-1: *Schematic example of WPW. Known as the great “mimic” — the ECG picture of WPW may resemble infarction, ischemia, chamber enlargement and/or bundle branch block — as it does here (See text).*

05.39 – FIGURE 05.39-1: *Recognizing WPW on a 12-Lead*

Primary care care and emergency clinicians will *not* always have the luxury of a baseline 12-lead ECG at the time a patient with *WPW-associated* tachycardia is initially seen. However, with luck — a ***baseline 12-lead ECG*** with ***telltale features*** of WPW may occasionally be found in the patient’s chart — thereby *confirming* the diagnosis. We show such a tracing with ***overt WPW*** in **Figure 05-39-1**. Note the following:

- Suggestion of **WPW** is readily apparent from initial inspection of **lead II** — in which all 3 ECG features are seen: **i)** short PR interval; **ii)** *delta* wave (red arrow); and QRS widening. The diagnosis of WPW is *confirmed* by recognition of *definite* delta waves in *other* leads (*III, aVF; V4, V5, V6*).
- **Negative delta waves** are seen in **leads aVR and aVL** (*blue arrows*). The negative delta wave in lead aVL may simulate high lateral infarction.
- **NOTE:** Delta waves are *not* prominent in every lead. The delta wave is subtle in leads I and V2 — and completely *absent* in lead V1. The QRS complex does *not* appear wide in these leads. Depending on the *relative* contribution from normal AV nodal pathway conduction and AP (*Accessory Pathway*) conduction — more or less leads may manifest delta waves, and the QRS complex may be obviously wide or only minimally prolonged.
- **Clinically** — One can *not* diagnose LVH, ischemia or infarction from the ECG shown in Figure

05.39-1 — *despite* the tall inferior R waves, deep Q wave in lead aVL, and ST-T wave changes in V1,V2,V3 — since the patient has WPW.

Bottom Line: It should be apparent from Figure 05.39-1 that the diagnosis of WPW could be *easily* overlooked IF one was not systematic in their approach. At *first* glance — the QRS complex does *not* look overly wide. That said — there is no mistaking the **lead II** findings of a **short PR interval** with **upward delta wave** (red arrow) — that when *added* to the remaining portion of the QRS results in **QRS widening**. Confirmation of WPW is forthcoming from recognition of delta waves in most (*but not all*) of the *other* leads on the tracing.

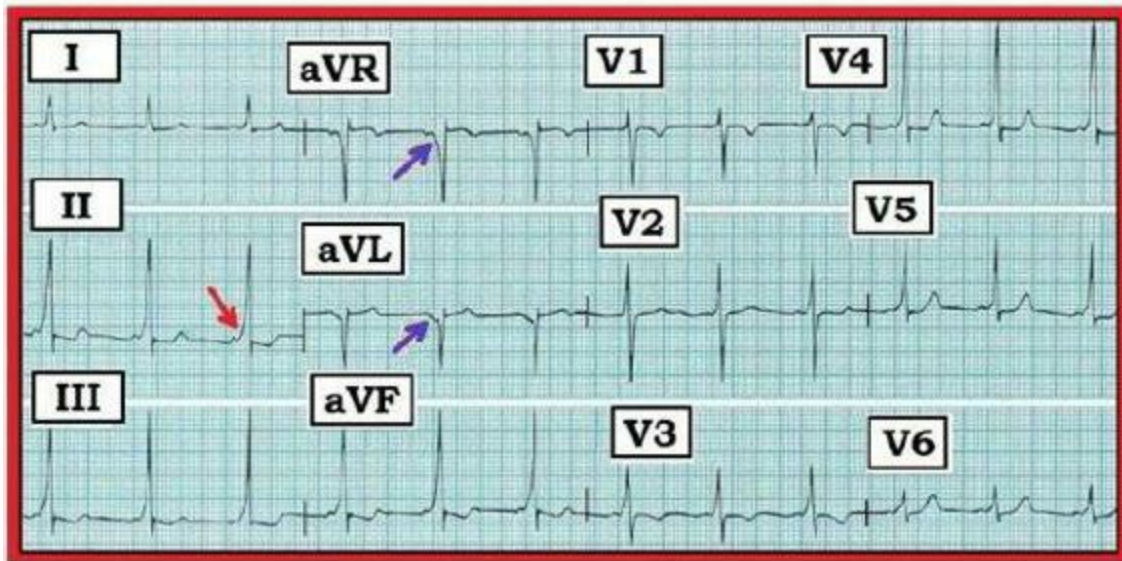


Figure 05.39-1: 12-lead ECG showing sinus rhythm with WPW. Note the short PR, delta wave and QRS widening in **lead II** (red arrow). Delta waves are *negative* in leads aVR and aVL (blue arrows). Delta waves are subtle in leads I,V2 and no delta wave at all is seen in lead V1 (*See text*).

05.40 – FIGURE 05.40-1: *Recognizing WPW*

Another 12-lead ECG example of WPW is provided below in Figure 05.40-1. It would be easy to misdiagnose this tracing as showing LVH *and/or* anterior infarction — IF one failed to recognize the *short* PR interval with *delta* waves in many leads (red arrows).

- The purpose of our ***sequential* Systematic Approach** to ECG interpretation — is to *avoid* overlooking entities such as WPW that is *easy* to do if your attention jumps first to QRS morphology *before* assessing intervals. As emphasized in our **ECG Crib Sheet** (*Section 00.6.3*) — the **3 Intervals** (PR/QRS/QT) should be assessed *early* in the process. Doing so should alert you to the *short* PR interval and *wide* QRS complex in Figure 05.40-1. We need to determine the **cause** of these **abnormal intervals** *before* proceeding further. *Knowing* the patient has WPW tells us that assessment for infarction, ischemia, conduction defects and chamber enlargement will *not* be reliable.

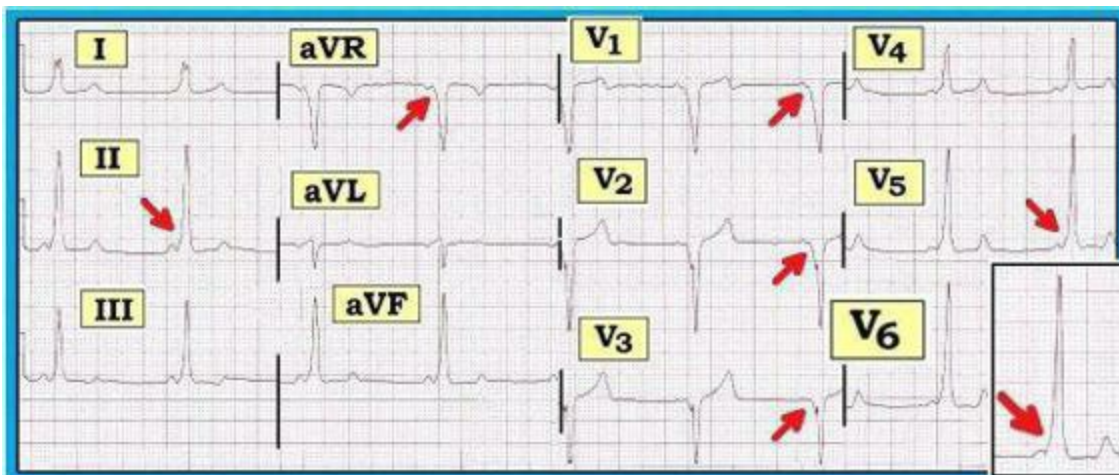


Figure 05.40-1: 12-lead ECG showing sinus rhythm with a *short* PR interval. The QRS is wide and *delta* waves are present in multiple leads (*arrows*). Note *negative* *delta* waves in V1,V2,V3 *simulate* anterior infarction. Increased QRS amplitude in V5,V6 *simulates* LVH. QRS morphology in leads I,V1,V6 *simulates* LBBB. This is **WPW**.

05.41 – FIGURE 05.41-1: *Atypical RBBB or WPW?*

We present one more 12-lead ECG to illustrate the challenge of distinguishing between bundle branch block and WPW (**Figure 05.41-1**).

- Doesn't the upright and widened QRS complex in lead V1 of **Figure 05.41-1** resemble RBBB?

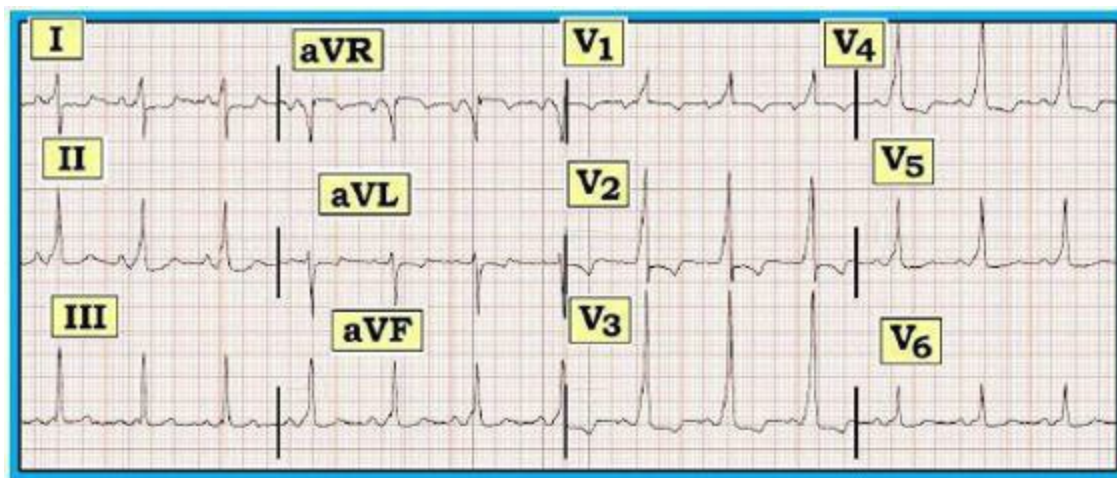


Figure 05.41-1: QRS widening with an upright QRS complex in lead V1. What is this due to? (See text).

Answer to Figure 05.41-1: As stated — there is obvious QRS widening in this tracing. Approaching this ECG systematically — the QRS complex in lead II appears to begin *very soon* after completion of the P wave in this lead. Admittedly — the PR interval does not seem overly short in every lead on this tracing. However, *suspicion* of a *short* PR interval should alert you to look closer at the QRS complex:

- Slurring on the upstroke of the *initial* part of the QRS complex is present in a number of leads (*leads II, aVF, and V1-thru-V6*). These are **delta waves** — and they confirm that the reason for QRS widening is **WPW**. There is no RBBB.
- As we will see in Section 10 when we discuss **LIST #6** (*Section 10.47*) — WPW is one of the important causes of a tall R wave in lead V1.



WPW: Localizing the AP

Beyond-the-Core: Delta wave morphology and orientation on 12-lead ECG may assist in surprisingly *accurate* localization of the AP (*Accessory Pathway*) in the patient with WPW. This is of more than academic interest to the EP cardiologist — as it facilitates and expedites localization of the AP during EP (*ElectroPhysiology*) study. In addition — it helps in planning the procedure as well as in patient discussion, since risks of catheter ablation and likely success rates are based in part on localization of the AP.

- We emphasize that ECG localization of the AP is an **advanced topic** that clearly extends beyond clinical needs of the non-EP-cardiologist. Practically speaking — it suffices to recognize WPW and IF there is need for referral. That said — ECG localization of the AP is a fascinating topic that is not necessarily difficult if certain basic parameters are followed.
- ***The choice is YOURS!*** Feel free to *SKIP OVER* this **Addendum #1** on *localizing* the AP with WPW (*Sections 05.42-thru-05.46*) — if you prefer *not* to get into this aspect of ECG interpretation. I avoided this topic for the first 30 years of my academic career — and my ECG interpretation ability was none the worse for it. BUT if you'd like to be “let in” on a most *user-friendly* approach I've encountered for AP localization — *Read on!* I wager you'll be *pleasantly* surprised at how *easy* approximate AP localization can be from a few well spent seconds analyzing the 12-lead ECG using the *step-by-step* approach I outline below (*Clinical examples discussed in Sections 05.44, 05.45, and 05.46*).

My **Suggested Approach** is based on my synthesis of material primarily from the following 2 references:

- Das MK, Zipes DP: *Electrocardiography of Arrhythmias — A Comprehensive Review* (ePub book). Elsevier-Saunders, Philadelphia, 2012.
- Fitzpatrick AP Gonzales RP Scheinman MM, et al: *Algorithm for the Localization of Accessory Atrioventricular Connections Using a Baseline Electrocardiogram*. J Am Coll Cardiol 23:107-116, 1994.

05.43 – WPW: *The Basics of AP Localization*

ECG localization of the AP is *not* perfect. Accuracy of ECG localization is clearly *suboptimal* if there is less than maximum preexcitation — as may occur when the QRS is minimally widened because a substantial portion of ventricular activation is occurring by transmission of the impulse over the normal AV nodal pathway.

- ***Approximate distribution of AP sites*** is the following: **i)** Lateral (*Left Ventricular*) Free Wall — ~50% of APs; **ii)** Right or Left *PosteroSeptal* Area — ~20%; **iii)** RV (*Right Ventricular*)

Free Wall — ~20%; and iv) *AnteroSeptal* Area — ~10%.

- On occasion — *more* than a single AP may be present in a given patient. Together with variation in the *relative* amount of AP vs normal pathway conduction — this may account for *changing* delta wave morphology that can sometimes be seen from one ECG to the next in a given patient.
- As a general rule — IF the **delta wave** (*first ~40msec of the QRS*) is **upright** (*positive*) in lead **V1** (*RBBB pattern*) — then there is a **LEFT-sided AP** (*as in Step A-1*).
- IF the **delta wave** is **downward** (*negative*) in lead **V1** (*LBBB pattern*) — then as a general rule there is a **RIGHT-sided AP** (*The major exception to this is when transition occurs between leads V1-to-V2 — as described in Step B-1*).

Step A-1: IF the QRS in Lead V1 is UPRIGHT

When the QRS complex is *upright* in lead V1 — then there is a “RBBB pattern”. In this case — **transition** (*where the R wave becomes taller than the S wave*) is said to occur before or by lead V1. This defines a **LEFT-sided AP**. Proceed as follows:

- **Measure** the **sum** of **delta wave polarities** in the **3 inferior leads** (*II, III, aVF*) — giving a score of +1 if the delta (*first 40msec of the QRS*) is positive; 0 if the delta is isoelectric; and -1 if the delta is negative.
- IF Sum of *inferior* lead Polarities is +2 or +3 = **AnteroLateral LV Free Wall AP**.
- IF Sum of *inferior* lead Polarities is *less* than +2 — then the AP is *posterior*.
- IF Sum of *inferior* lead Polarities is -2 or -1 — and — the R wave in lead I is *at least* 0.8mV (8 mm) *more* than the S wave in lead I = **PosteroSeptal AP**. Otherwise = there is a **PosteroLateral LV Free Wall AP**.

Step A-2: IF Transition Occurs Between Lead V1-to-V2:

IF the R wave in lead V1 is *less* than the S wave in V1 — but by lead V2 the R wave *becomes* taller than the S wave in V2 (ie, *IF transition occurs between V1-to-V2*) — then the AP could be either left or right-sided. Proceed as follows:

- IF the R wave in lead I is less than 1.0mV (10 mm) greater than the S wave in lead I = then there is a **LEFT-sided AP**. In this case — Proceed as above (**Step A-1**) for when the QRS is UPRIGHT (*beginning by measuring sum of delta polarity in the inferior leads to determine if the AP is anterolateral — posteroseptal — or posterolateral*).
- But IF the R wave in lead I is *at least* 1.0mV (10 mm) more than the S wave in lead I = then there is a **RIGHT-sided AP**. In this case — Proceed as described in **Step B-1**.

Step B-1: How to Tell IF the AP is RIGHT-sided?

When the QRS complex is downward (*negative*) in lead V1 — and — **transition** (*where the R wave becomes taller than the S wave*) occurs after lead V2 — then there is a **RIGHT-sided AP**.

- As stated above there can also be a **RIGHT-sided AP** — IF transition occurs *between* V1-to-V2 — and — the R wave in lead I is *at least* 1.0mV (10 mm) more than the S wave in lead I.

Step B-2: When the AP is RIGHT-sided:

Localization of a *right-sided* AP will depend on **where transition occurs**. There are **3 possibilities**. Transition may be: **i) by or before V2-to-V3 (Step B-3)**; **ii) between V3-to-V4 (Step B-4)**; or **iii) after V4 (Step B-5)**.

Step B-3: Right-Sided AP: Transition by or before V2-to-V3:

IF the AP is *right-sided* with transition by or before lead V2-to-V3 — then the AP is **Septal**. To determine *which* septal area is involved — Proceed as follows:

- Measure the **sum of delta wave polarities** in the **3 inferior leads (II, III, aVF)** — giving a score of +1 if the delta (*first 40msec of the QRS*) is positive; 0 if the delta is isoelectric; and -1 if the delta is negative.
- IF Sum of *inferior* lead Polarities is -2 or -3 = ***PosteroSeptal AP***.
- IF Sum of *inferior* lead Polarities is -1, 0 or +1 = ***MidSeptal AP***.
- IF Sum of *inferior* lead Polarities is +2 or +3 = ***AnteroSeptal AP***.

Step B-4: Right-Sided AP: Transition between V3-to-V4:

IF the AP is *right-sided* with transition between lead V3-to-V4 — then the AP is either septal or right ventricular free wall. To determine *which* — Proceed as follows:

- Measure **delta wave amplitude** (*first ~40msec of the QRS*) in **lead II**.
- IF the delta wave in lead II is *at least* 1.0mV (10mm) = ***Septal AP***. To then find out *which* septal area is involved — Proceed as above for when transition is between V2-to-V3 (Step B-3).
- IF the delta wave in lead II is *less* than 1.0mV (10mm) = ***RV Free Wall AP***. To determine IF the AP is located on the *anterolateral* or *posterolateral* RV Free Wall — Proceed as below for when transition is after V4 (Step B-5).

Step B-5: Right-Sided AP: Transition after Lead V4:

IF the AP is *right-sided* with transition after lead V4 — then the AP is located in the ***RV Free Wall***. To determine IF the AP is located on the *anterolateral* or *posterolateral* RV Free Wall — Proceed as follows:

- Measure the **delta wave frontal axis** (*looking at delta wave polarity in leads I and aVF*).
- IF the delta wave frontal axis is positive (= *more than 0 degrees*) = ***Anterolateral RV Free Wall AP***.
- But IF the delta wave frontal axis is negative (= *less than 0 degrees*) — then look at the R wave in lead III.
- IF delta wave frontal axis is *negative* — and — the R wave in lead III is *net positive* = ***AnteroLateral RV Free Wall AP***.
- IF delta wave frontal axis is *negative* — and — the R wave in lead III is *net negative* = ***PosteroLateral RV Free Wall AP***.

05.44 – FIGURE 05.44-1: *Where is the AP?*

Let's apply the above approach for AP localization to the ECG shown in **Figure 05.44-1** (*this ECG was previously seen in Figure 05.40*). *Where is the AP likely to be?*

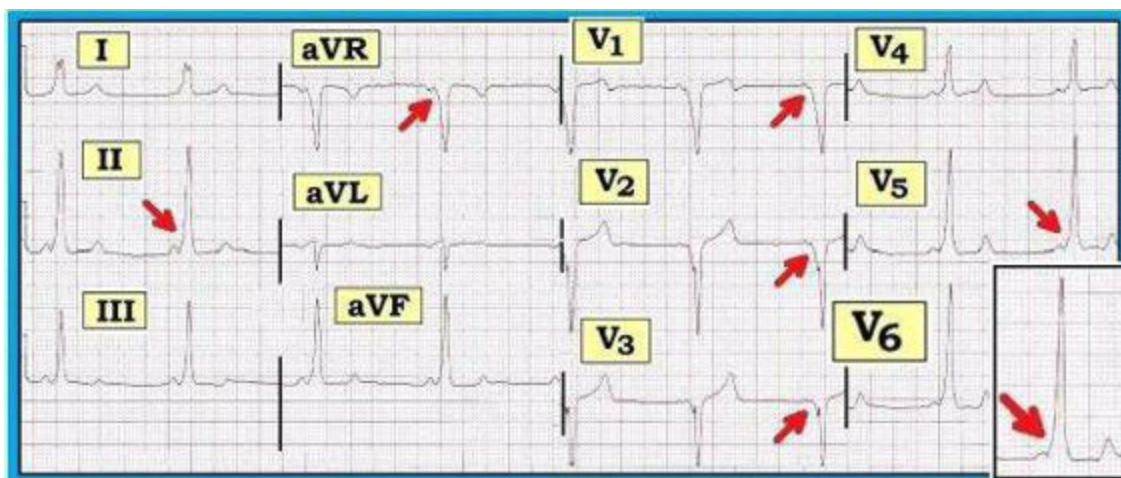


Figure 05.44-1: Sinus rhythm with WPW. *Where is the AP?*

Answer to Figure 05.44-1: We begin by looking to see IF the QRS complex in lead V1 of this tracing is upright or negative:

- Since the QRS in V1 is negative — we *skip* over Step A-1.
- **Transition** (*where the R wave in precordial leads becomes taller than the S wave*) is not between V1-to-V2 — therefore we also skip over Step A-2.
- According to **Step B-1** — the AP is ***RIGHT-sided*** (*because the QRS is negative in V1 and transition occurs after lead V2*).
- **Transition** occurs *between V3-to-V4*. Therefore we skip to **Step B-4**. We are asked to measure ***delta wave amplitude*** in lead II. Realizing that it is *not* always easy to distinguish the precise end of slurring from the delta wave vs the point of transition to the remaining portion of the QRS — it looks like there is a *markedly* positive delta wave (*of at least 10mm*) in lead II. This suggests a ***Septal*** location for the AP.
- To determine the likely part of the septum that is involved — We are asked to return to **Step B-3**. Delta wave polarities are clearly positive in *each* of the inferior leads — therefore we suspect an ***AnteroSeptal*** AP location.

05.45 – FIGURE 05.45-1: *Where is the AP?*

Localize the AP for the example of WPW shown below in **Figure 05.45-1** (*this ECG was previously seen in Figure 05.41-1*). *Where is the AP likely to be?*

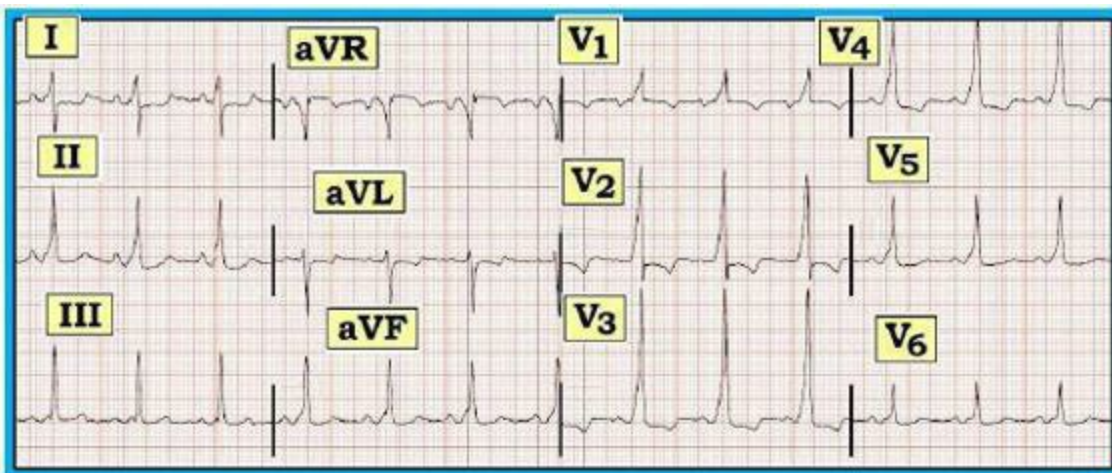


Figure 05.45-1: Sinus rhythm with WPW. *Where is the AP?*

Answer to Figure 05.45-1: We begin again by looking to see IF the QRS complex in lead V1 of Fig. 05.45-1 is upright or negative:

- Since the QRS complex in lead V1 is upright — we begin with **Step A-1**. Because the QRS is positive in lead V1 — we already know we are dealing with a **LEFT-sided AP**.
- The sum of **delta wave polarities** is *at least +2 (decidedly positive delta waves in leads II, aVF — though no more than minimally positive in III, if not isoelectric in this lead)*. Therefore — we suspect an **AnteroLateral LV Free Wall AP**.

05.46 – FIGURE 05.46-1: *Where is the AP?*

Localize the AP for the example of WPW shown below in **Figure 05.46-1** (*this ECG was previously seen in Figure 05.39-1*). *Where is the AP likely to be?*

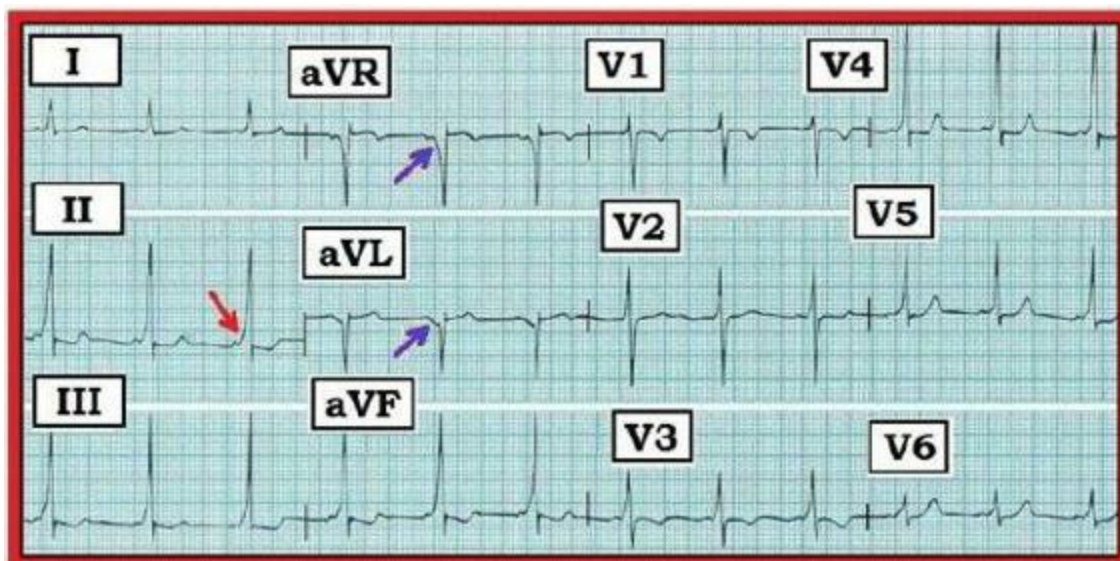


Figure 05.46-1: Sinus rhythm with WPW. *Where is the AP?*

Answer to Figure 05.46-1: We begin again by looking to see IF the QRS complex in lead V1 of Fig. 05.46-1 is upright or negative:

- Since the QRS complex in V1 is negative — we *skip* over Step A-1.
- **Transition** (*where the R wave in precordial leads becomes taller than the S wave*) is not between V1-to-V2 — therefore we also skip over Step A-2.
- According to **Step B-1** — the AP is **RIGHT-sided** (*because the QRS is negative in V1 and transition occurs after lead V2*).
- **Transition** occurs *between V2-to-V3*. Therefore we skip to **Step B-3**. We are asked to measure the sum of ***delta wave polarities*** in the inferior leads. Given that delta wave polarities are decidedly positive in *each* of the inferior leads — we suspect an **AnteroSeptal** AP location.

05.47 – Addendum #2: *Arrhythmias with WPW*



We have already emphasized how conduction of the sinus impulse in patients with WPW may be: **i)** via the normal (*AV nodal*) pathway; **ii)** down the AP (*Accessory Pathway*); or **iii)** it may *alternate* between the two. The *same* 3 possibilities for conduction exist when a patient with WPW develops a **supraventricular tachyarrhythmia**. In these next few sections — we briefly review key points to consider when the patient with WPW develops an SVT (*SupraVentricular Tachycardia*).

- Patients with WPW are prone to SVT rhythms in which a **reentry circuit** is set up between the normal AV nodal pathway and the AP. Assuming there is no preexisting bundle branch block — whether or not the QRS complex will be wide *during* the tachycardia in a patient with WPW will depend upon whether the reentrant pathway goes *up* or *down* the AP (**Figure 05.47-1**).

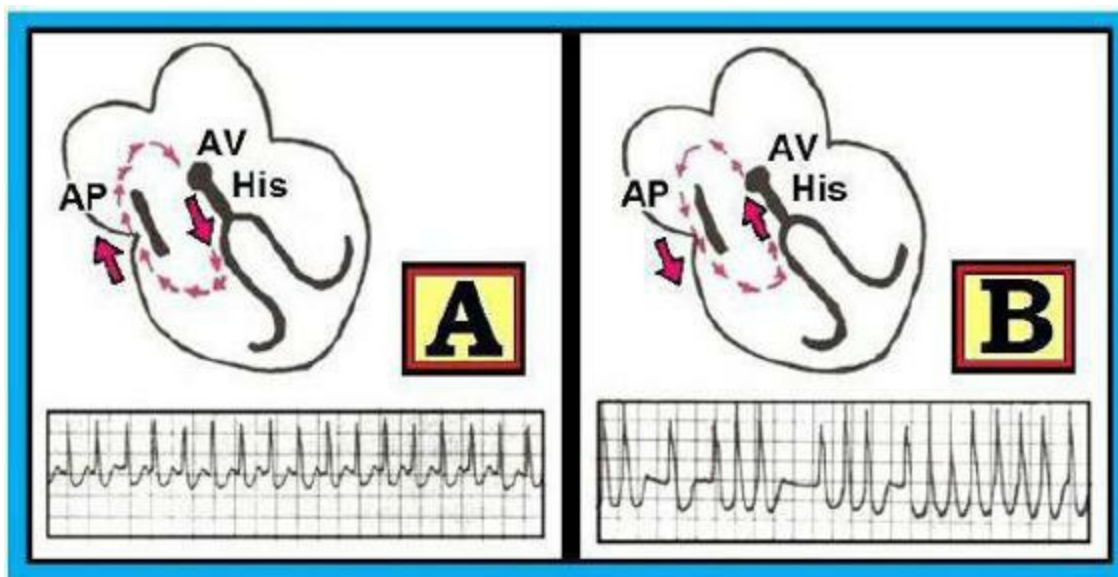


Figure 05.47-1: SVT pathways with WPW. Conduction of the impulse from atria to ventricles during *WPW-associated* tachycardia may either be: **i)** as in **Panel A** = **orthodromic** (*down the normal AV nodal-His-Purkinje system — and back up the AP*) — as commonly occurs with PSVT; or **ii)** as in **Panel B** = **antidromic** (*first down the AP — and then back up the normal pathway*) — as commonly occurs with AFib or AFlutter — and only rarely with PSVT. Assuming there is no underlying bundle branch block — the QRS will be *narrow* with orthodromic conduction (**Panel A**) and *wide* with antidromic conduction (**Panel B**).

05.48 – PSVT with WPW: *When the QRS During Tachycardia is Narrow*

When PSVT occurs in a patient with WPW — the tachycardia is almost always **orthodromic** (*down the normal AV nodal-His-Purkinje system — and back up the AP* = **Panel A** in **Figure 05.47-1**).

- Because conduction goes *down* the normal AV nodal pathway — the **QRS** is **narrow** during the tachycardia. As a result — the usual **AV nodal blocking drugs** (*Verapamil-Diltiazem-β-*

Blockers) can be used and are usually effective.

- A delta wave will *not* be seen during the tachycardia. The presence of WPW may only be suspected in a patient with *narrow-complex* PSVT IF an ECG showing delta waves is found in the medical chart or obtained following conversion of the tachycardia.
- PSVT is by far the most common tachyarrhythmia observed in patients with WPW. It is often well tolerated.
- *Beyond-the-Core*: A surprising number of patients with PSVT actually have one or more *concealed* accessory pathways. That is — a conduction pathway exists between atria and ventricles that *only* allows orthodromic (*but not antidromic*) conduction. Since forward conduction down the AP is not possible — a delta wave is *never* seen. However, ready availability of an AP reentry pathway may predispose such patients to frequent episodes of PSVT. While acute treatment considerations are similar to those for treatment of any other *narrow-complex* PSVT — awareness of this entity may lower one's threshold for EP referral after the episode if PSVT episodes are frequent *and/or* difficult to control with medication. Ablation of the AP may be curative.
- *Way-Beyond-the-Core*: Taking the last *advanced* information bullet one step further — You may at times be able to *suspect* the presence of an AP in some WPW patients with *narrow* complex PSVT even without seeing a delta wave IF you see a *negative* P wave with *long* R-P interval reflecting *retrograde* conduction back to the atria during the reentrant cycle. When retrograde conduction is seen during AVNRT in a patient *without* WPW — the RP is *very* short (*most often seen as a notch at the tail end of the QRS complex*) reflecting short distance travel *within* the AV node. The RP tends to be longer (*negative P usually seen midway within the ST segment*) for a patient in whom the reentry circuit runs down the AV node and back up an AP lying outside the AV node. Technically — this type of PSVT in a patient with accessory pathways is known as **AVRT** (*AtrioVentricular Reciprocating Tachycardia*). Distinction in the ECG picture *between* AVNRT vs AVRT (*when an AP is present*) is an *advanced* concept that generally does *not* impact on acute treatment. We often will simply *not* know if a *narrow-complex* PSVT rhythm involves AV node vs AP in its reentry circuit.
- *Final Way-Beyond-the-Core Point*: Adenosine may *shorten* the refractory period of atrial tissue — which could initiate AFib in a *predisposed* individual. As a result — Adenosine should be used with caution in patients with *known* WPW, given *theoretic* possibility of inducing AFib (*which could have significant consequence in a patient with accessory pathways*). That said — we often will *not* know at the time treatment is needed IF the patient with a *narrow-complex* PSVT has a *concealed* AP and even if they do, giving Adenosine to such a patient will *rarely* be problematic.

BOTTOM Line = What to Remember!

Most of the time — PSVT in a patient with WPW will conduct with a *narrow* QRS complex (*as in Panel A in Figure 05.47-1*).

- Practically speaking — you do *not* have to worry in the immediate *acute* setting IF a patient with *narrow-complex* PSVT has WPW or not. Initial treatment measures are the same. These include:
i) A *vagal* maneuver; and/or **ii)** Adenosine; and/or **iii)** *Other* AV nodal blocking agent (*Diltiazem, β -Blocker*).

- With regard to *longterm* management — awareness that a significant percentage of patients with *narrow-complex* PSVT have a *concealed* AP (*even when they never show overt WPW on their ECG*) should *lower* our threshold for EP referral if PSVT episodes recur *despite* medical therapy.

05.49 – *Very Rapid AFib with WPW*

In contrast to the situation for PSVT with WPW — the occurrence of **AFib** in a patient with WPW *almost always* manifests a **wide QRS** during the tachycardia. This is because the path of conduction for AFib with WPW is almost always **antidromic** (*first down the AP — and then back up the normal pathway* = **Panel B** in Figure 05.47-1).

- Because of the short RP (*Refractory Period*) of the accessory pathway — there may be 1:1 conduction of atrial impulses (*at times resulting in a ventricular response that may exceed 250/minute!*). As might be anticipated — these rapid rates are not always well tolerated (*may deteriorate to VFib*).
- It is recognition of the ECG picture of **exceedingly rapid AFib** (*over 220/minute in parts of the tracing*) in conjunction with **QRS widening** and **marked variability** in regularity of the tracing that clues the clinician into the *almost certain* diagnosis of **WPW with very rapid AFib**. This is the case for the rhythm in Figure 05.49-1.

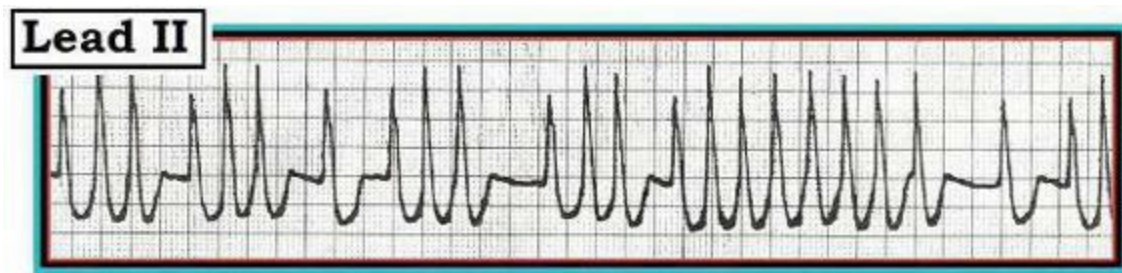


Figure 05.49-1: WPW with *very rapid* AFib. Even *without* benefit of a 12-lead ECG — an *almost certain* diagnosis of **WPW** can be made from this *single* rhythm strip because: **i)** there is QRS widening with *marked* irregular irregularity showing far *more* variation in rate than is seen with VT; **and ii)** parts of the rhythm show a rate *between* 250-300/minute, which is far *too fast* for AFib conduction over the normal AV nodal pathway. Thus, the rhythm *must be* AFib — **and** conduction must be *bypassing* the normal AV nodal pathway in a patient with WPW who has an AP (*Accessory Pathway*).

Treatment Considerations: WPW with Very Rapid AFib

The importance of recognizing that the example of *very rapid* AFib seen in Figure 05.49-1 is from a patient with WPW — is that treatment considerations are very *different* than they are for the much more common usual AFib patient.

- **AV Nodal Blocking Drugs** — that are regularly used to treat the common form of rapid AFib are **contraindicated**. This includes Verapamil-Diltiazem-Digoxin — and *possibly* β -Blockers.

By impeding conduction down the normal AV nodal pathway — *all* of these agents may inadvertently *facilitate* forward (*antidromic*) conduction of AFib impulses down the AP (*Accessory Pathway*), thereby *accelerating* the rapid AFib even more. This may precipitate deterioration to VFib.

- Realizing that Adenosine is often used as a *diagnostic* measure during assessment of various WCT (*Wide-Complex Tachycardia*) rhythms — it is best to **avoid Adenosine** whenever possible IF very rapid AFib with WPW is suspected (*since Adenosine may likewise accelerate AP conduction in a patient with WPW*). That said — the *ultra-short* half-life of Adenosine is much *less* likely to be deleterious compared to other AV nodal blocking drugs if it is inadvertently given.
- **Drugs of Choice:** The 3 drugs that have most commonly been recommended for antiarrhythmic treatment of **hemodynamically stable** very rapid AFib (*or AFlutter*) with WPW are **i)** Procainamide; **ii)** Amiodarone; and **iii)** Ibutilide. There are pros and cons for use of each of these agents that extend beyond the scope of this ePub on ECG interpretation. Each drug has its advocates. All in theory reduce forward transmission of impulses down the accessory pathway. **KEY Point:** IF at any time during the treatment process the patient becomes *hemodynamically* unstable — then cardiovert!

05.50 – Atrial Flutter with WPW

When **AFlutter** occurs in a patient with WPW — the tachyarrhythmia is also **antidromic** (*first down the AP — and then back up the normal pathway = as in **Panel B** of Figure 05.47-1*).

- As with AFib — the **QRS** is **wide** in **AFlutter** with **WPW**. There may be 1:1 AV conduction of atrial impulses (*so that the ventricular response may be 250-300/minute*)!
- Very fast AFlutter with WPW is seen even *less often* than AFib (*but clinical manifestations and treatment are similar*).

05.51 – PSVT with WPW: *When the QRS is Wide*

In *rare* instances — PSVT may be **antidromic** (ie, *travel first down the AP — and then back up the normal pathway = as occurs in **Panel B** of Figure 05.47-1*).

- In these rare instances — the **QRS** will be **wide** and the PSVT rhythm may be *indistinguishable* from VT.
- It may only be *after* conversion to sinus rhythm that “telltale” delta waves of WPW can be identified. Fortunately — the *vast* majority (~95%) of PSVT episodes with WPW are orthodromic (*with narrow QRS*). **Synchronized cardioversion** will be the usual treatment of choice for episodes of a *regular* WCT (*Wide-Complex Tachycardia*) in which one suspects *antidromic* PSVT in a patient with WPW as the etiology.

05.52 – FIGURE 05.52-1: *VT or WPW? What to Do?*

We complete our discussion of arrhythmias in the patient with WPW by the 12-lead ECG shown in **Figure 05.52-1** — obtained from a *hemodynamically* stable young adult who presented to the ED (*Emergency Department*) with *new-onset* palpitations.

- *What is the rhythm?* What does this patient have?
- How should the patient be treated?
- Which drugs should not be given?

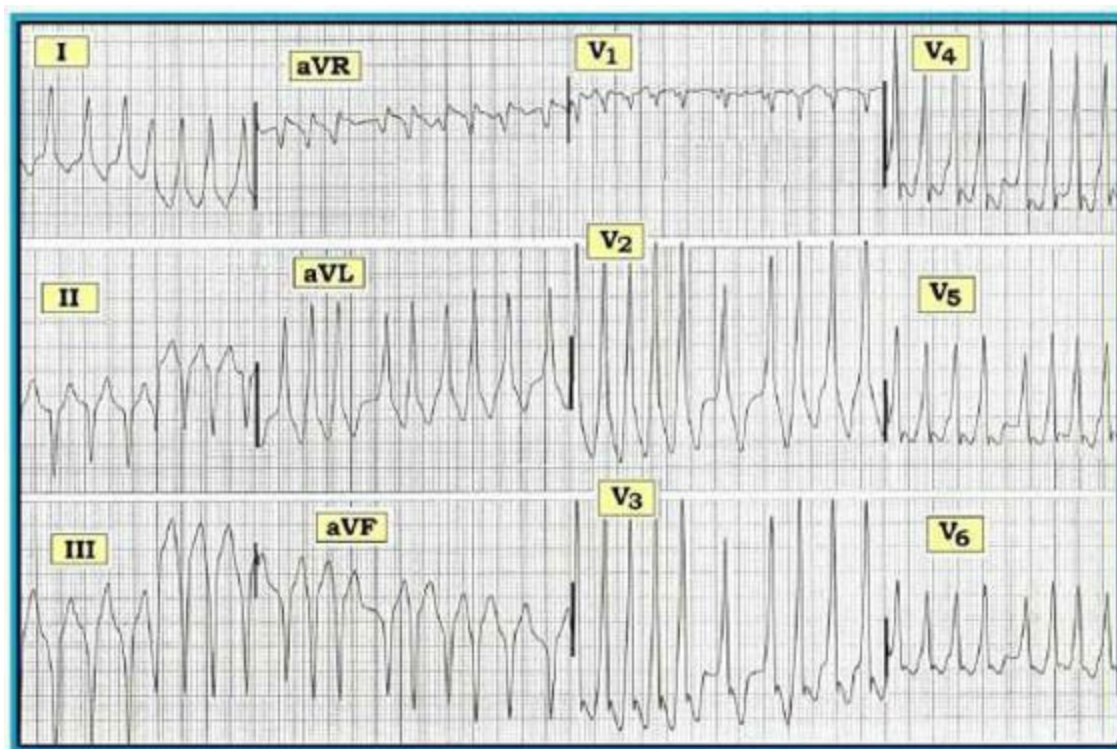


Figure 05.52-1: 12-lead ECG from a young adult with palpitations. What is the rhythm? (*See text*).

Answer to Figure 05.52-1: The rhythm in this *hemodynamically* stable young adult is an **irregularly irregular WCT** (*Wide-Complex Tachycardia*). No P waves are seen in any of the 12 leads of this tracing. This defines the rhythm as **AFib**. That said — the **ventricular response** is **exceedingly rapid** (*attaining a rate of nearly 300/minute in some parts of the tracing*). In addition — there is marked **variability** in rate of the ventricular response (*seen best in leads aVF and V3*).

- This 12-lead ECG is **virtually diagnostic** of **very rapid AFib** in a patient who has **WPW**. VT (*Ventricular Tachycardia*) may at times be *slightly* irregular — but it should not be as *irregularly* irregular as seen here *throughout* the tracing.
- The *rapidity* of the rate (*nearly 300/minute in certain parts of the tracing*) — suggests AP conduction until proven otherwise (*Section 05.49*). Treatment considerations are as described in *Section 05.49* (*IV Amiodarone, Procainamide or Ibutilide if the patient remains stable — with immediate cardioversion at the first sign of instability*).
- The patient should be referred to an EP cardiologist after resolution of the acute tachycardia for consideration of an ablative procedure that may be curative.

The QT Interval/Torsades

06.1 – How to Measure the QT

The **QT interval** — is the period that extends from the *beginning* of ventricular depolarization — until the *end* of ventricular repolarization (**Figure 06.1-1**). For practical purposes, the QT is **prolonged** — IF it clearly measures **more** than **half** the **R-R interval**. The **principal exception** to this rule is when the heart rate is rapid (>90 - 100 /minute) — in which case it becomes more difficult to measure the QT and determine its clinical significance.

- **To Measure the QT** — Select a lead where you can clearly see the *end* of the T wave. Select *that lead* in which the QT appears to be *longest*.
- **NOTE:** If there is no “q” wave in the QRS complex — Measure from the beginning of the R wave (**Figure 06.1-1**).

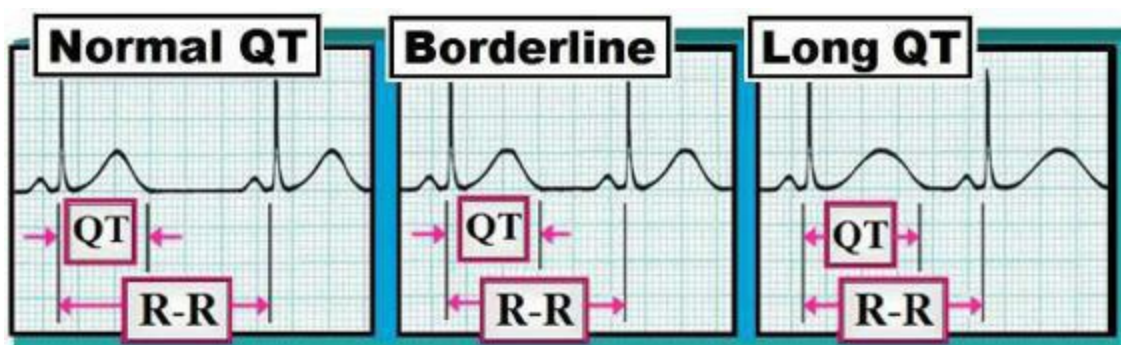


Figure 06.1-1: Provided that heart rate is *not* overly fast (ie, 90 - 100 /minute) — the **QT** is **normal** if it is *not more* than half the R-R interval. There are 3 possibilities: i) **Left Panel** — The **QT** is clearly **normal**; ii) **Middle** — The **QT** is “**borderline**”, as it is approximately *half* the R-R interval (or at most, *slightly more than half* the R-R interval); or iii) **Right** — The **QT** is **clearly prolonged**.

06.2 – LIST #3: Causes of QT Prolongation

Clinically — We want to know IF the QT is normal or long. This is usually easy to tell by the “**eyeball**” method (ie, *Is the QT more than half the R-R interval?*) — provided that the heart rate is *not* excessively fast (>90 - 100 /minute).

- Practically speaking — one only cares IF the QT interval is normal, borderline, or long. Patients with a long QT interval are at *increased* risk of developing the potentially *life-threatening* arrhythmia Torsades de Pointes (*Section 06.6*).
- **Beyond-the-Core:** **Hypercalcemia** produces **QT shortening**. That said — Hypercalcemia is *difficult* to recognize on ECG, and is usually *only* seen with very high serum calcium values (of >12 mg/dL).

Causes of a Long QT:

The common causes of a *long* QT interval can be divided into 3 categories: **i)** Drugs; **ii)** Electrolyte disorders; and **iii)** CNS catastrophes. We consolidate this information in our **LIST #3** (Figure 06.2-1):

- **KEY Point:** The NOTE at the bottom of List #3 reminds us that *other* conditions (ie, *ischemia/infarction/BBB*) — may also prolong the QT. Clinically — We pay *less* attention to the QT interval when the ECG picture is dominated by *other* findings. But IF the *only* thing wrong (*beyond nonspecific changes*) is a long QT — Think “**Drugs/Lytes/CNS**” as the cause!

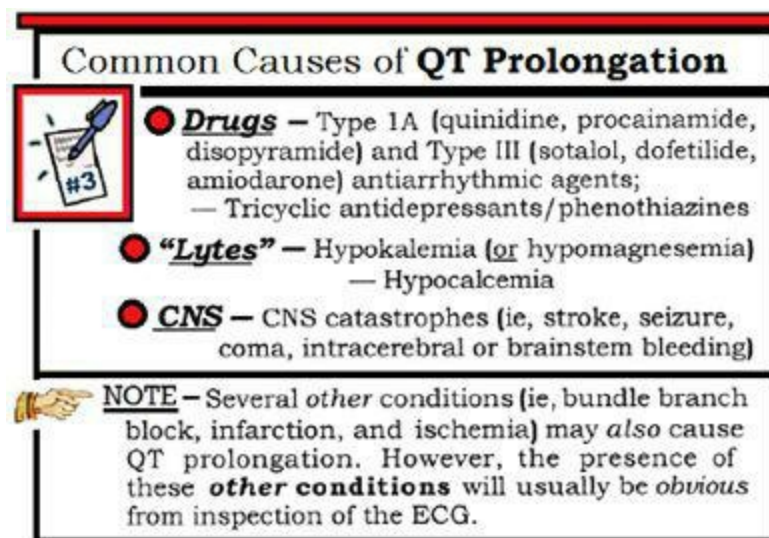


Figure 06.2-1: List #3 = Common Causes of a *Long* QT Interval.

06.3 – A Closer Look at LIST #3: *Drugs – Lytes – CNS*

We expand on the causes of QT prolongation in LIST #3 (Figure 06.2-1) — by emphasizing the following points:

- **DRUGS** — An ever *increasing* number of drugs (*in addition to those in List #3*) may *either* affect the QT interval directly — *and/or* — produce p450 system drug interactions that increase Torsades risk if taken together with *other* QT-lengthening drugs. Clinically — it is usually *not* a problem in an otherwise healthy adult to take any single drug that may affect the QT. But — the amount of QT prolongation (*and the risk of Torsades*) may be *additive IF more* than one *QT-lengthening* drug is taken — especially if circumstances or *other* condition *predisposing* to Torsades is present (Section 06.4).
- **LYTES** — 3 electrolyte disorders should be kept in mind when assessing a patient with QT prolongation: **i) Hypokalemia** — is characterized by ST flattening/depression; development of **U waves**; and QT lengthening (Section 11.7); **ii) Hypomagnesemia** — produces *identical* ECG and clinical effects as hypokalemia (Section 11.8); and **iii) Hypocalcemia** — which if other electrolytes are normal, tends to produce a *normal* T wave after a *prolonged* QT interval (Section 11.1).
- **NOTE:** Rather than “QT” lengthening — it is really the “**QU**” **interval** that is prolonged with hypokalemia *and/or* hypomagnesemia (*as T and U waves tend to fuse as the U wave becomes*

bigger with more profound electrolyte depletion).

- **CNS catastrophes** (ie, *coma, stroke, trauma, seizure, bleed*) — are known to produce some of the most **bizarre ST-T wave abnormalities** (*and some of the longest QT intervals*). Resultant ST segment elevation that may be seen with CNS catastrophes can at times *mimic* the changes of *acute MI*. The reason for ST-T wave abnormalities with CNS disorders is *uncertain* — but is thought to relate to disturbance of autonomic tone.

06.4 – Conditions *Predisposing to a Long QT/Torsades*

Be aware of circumstances that *increase* the **risk** of QT prolongation and therefore of developing *Torsades de Pointes*. These include:

- **Female sex** (*women are more predisposed to Torsades!*).
- **Electrolyte disorders** (*low K⁺/low Mg⁺⁺*).
- **Bradycardia** (*usually 50/minute*) — since a long *preceding* R-R interval (*as seen with bradycardia*) sets up prolongation of the QT interval for the following beat.
- **Structural heart disease** (*heart failure; marked LVH; cardiomyopathy; ischemia; prior infarction*).
- Significant **renal** or **hepatic dysfunction**.
- **Baseline QT prolongation** — if the QTc is >450 msec. (*especially if >500 msec.* — Section 06.5).

06.5 – The QTc: *Corrected QT Interval*

Up to now — We have focused on the “*eyeball method*” for determining IF the QT interval is normal or long:

- IF the QT is less than *half* the R-R — then the QT is **normal**.
- But IF the QT is more than *half* the R-R — the QT is **long** (Figure 06.1-1).

In either case, more precise determination of the QT interval is usually not necessary — since for practical purposes, the main thing we are concerned with is whether the QT is normal or long. That said — at times it will be important to more precisely determine the QT. This is especially true when it is *not* readily apparent from inspection whether the QT interval is normal or long.

- Tables exist for precise determination of the QT based on the **age, sex** and **heart rate**. Accounting for difference in heart rate results in a **QTc** — where the “*little c*” indicates **correction** of the QT for heart rate.
- Beyond-the-Core: The formula for determining **QTc** = the QT that you measure — divided by the *square root* of the R-R interval. The “good news” is that at a heart rate of 60/minute — the R-R interval = 5 large boxes (= *1.0 second*) — and the square root of one is one! This means that at a rate of 60/minute — the QTc is the QT interval that you measure.

- **KEY Point:** The *computerized* interpretation will accurately calculate the QTc for you! In general — the **upper normal** for the QTc is **~450msec**. *More* than this suggests QT prolongation. *Definite* concern about QT prolongation increases once the QTc *exceeds* 480 msec (*especially if >500 msec*)!

06.6 – Torsades: *WHY Care about QT Prolongation?*

Why Care about the QT interval? The answer is to hopefully *prevent* the rhythm seen in **Figure 06.6-1**.

1. Cardiopulmonary resuscitation was ongoing at the time this tracing was obtained.

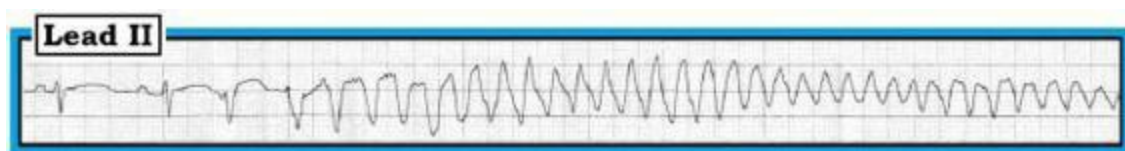


Figure 06.6-1: Irregular WCT rhythm obtained during cardiopulmonary resuscitation. What is the rhythm? What to do next? (*See text*).

Answer to Figure 06.6-1: After 2 sinus beats — the rhythm dramatically changes. This is **polymorphic VT** — as defined by constantly *varying* QRS morphology *throughout* the rest of the tracing.

- ***Torsades de Pointes*** — is defined as **polymorphic VT** that occurs in association **with** a **prolonged QT** interval on baseline ECG. The very rapid *irregular* WCT seen in **Figure 06.6-1** manifests the shifting QRS polarity around the baseline (“*twisting*” of the points) that is characteristic of Torsades.
- **NOTE:** There appears to be a **prolonged QT interval** for the first 2 sinus beats in **Figure 06.6-1** (ie, *the QT looks to be more than half the R-R interval*) — in which case the rhythm would be **Torsades**. That said — it is difficult to be certain where the T wave (*and therefore the “QT” interval*) ends in this tracing. Clinically — It will simply *not* always be possible to assess QT duration during *polymorphic VT*.
- ***Clinical Reality*** — When we *either* do not have access to a prior *baseline* 12-lead ECG on the patient or cannot accurately assess the QT interval from the rhythm strip — we will be *unable* to distinguish whether the rhythm is **PMVT** (**P**oly**M**orphic **V**entricular **T**achycardia) — or “**Torsades**” (*which is simply PMVT with baseline QT prolongation*).

Treatment of Figure 06.6-1: *Regardless* of whether this *irregular* WCT rhythm is PMVT or Torsades — **initial treatment measures** are the same: **i)** Defibrillation if the rhythm persists; **ii)** Magnesium Sulfate (1-2 gm IV — *which often needs to be repeated up to 4-8 gm*); **and iii)** Try to find and fix the *underlying* cause of PMVT/Torsades. Potential additional measures for resistant cases (ie, *overdrive pacing, isoproterenol*) extend beyond the scope of this ePub on ECG diagnosis.

- ***Beyond-the-Core:*** There are some differences in etiology and response to treatment between Torsades vs PMVT *without* QT prolongation that are worthy of mention. While IV Magnesium Sulfate is the drug of choice for *both* disorders — PMVT tends to respond less well to this

treatment than when there is Torsades *with* QT prolongation. Rather than being drug or electrolyte induced — PMVT with a *normal* QT more often has an *ischemic* etiology (occasionally due to *Brugada Syndrome*). Efforts addressed at treating acute ischemia may therefore be helpful. IV Amiodarone *and/or* β -blockers may reduce recurrence and should be considered if IV Magnesium is ineffective.

06.7 – FIGURE 06.7-1: Torsades vs PMVT vs Something Else?

Would you treat *both* of the rhythms in Figure 06.7-1 with defibrillation and IV Magnesium?

- What *additional* information is essential for answering this question?

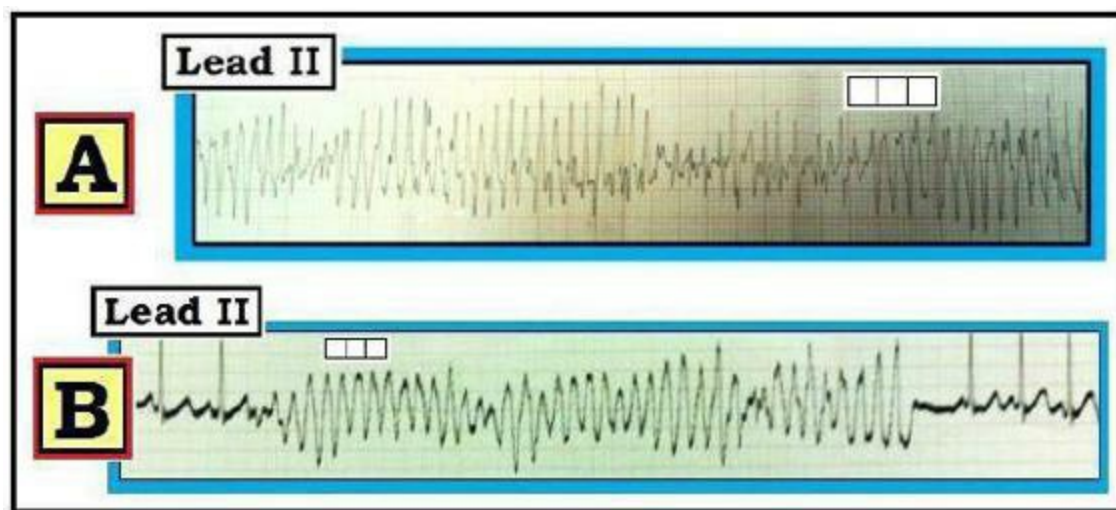


Figure 06.7-1: Torsades *vs* PMVT *vs* something else? (See text). HINT: Timelines for determining heart rate are shown by the *square* boxes in each tracing (corresponding to a large box on ECG grid paper in each case).

Answer to Figure 06.7-1: At first glance, it appears that *both* of these rhythm strips represent PMVT/Torsades. While superficially they do — the devil is in the details (*in this case the rate of the rhythm*).

- Note in **Rhythm A** of Fig. 06.7-1 — that there are at certain points in the tracing *two* vertical deflections for each large box. This corresponds to a rate of ~500-to-600/minute, or *too fast* for Torsades. This is **artifact**. We do *not* see any normal beats on this tracing — but instead just see very straight line, *excessively* fast deflections suggestive of artifact.
- The rate in **Rhythm B** is also fast — but much closer to ~300/minute (~1 complex per each large box) — which clearly falls within the appropriate rate range for **PMVT/Torsades**. The rhythm begins and ends by a few normal (*narrow*) sinus beats. Given the rapid rate — it is difficult to determine if the QT is or is not prolonged. Therefore, we can *not* be certain if **Rhythm B** is PMVT *or* technically qualifies as “Torsades”. As emphasized in Section 06.6 — initial treatment measures are the same *regardless* of whether or not there is baseline QT prolongation.

KEY Point: — Awareness of *clinical context* is an essential component of *clinical* ECG interpretation. The diagnostic dilemma of whether **Rhythm A** in Figure 06.7-1 is “real” might be *easily* resolved IF we *knew* that the patient was alert and hemodynamically stable (*which would confirm artifact*). Loose lead connections, tremor, scratching, shivering, and seizure activity are but a few of the possible causes of *artifact-related* deflections that might simulate a tachyarrhythmia.

06.8 – FIGURE 06.8-1: *Is the QT Long?*

Comment on the QT interval for the 12-lead ECG shown in Figure 06.8-1. What *clinical* conditions are likely to account for findings on this ECG?

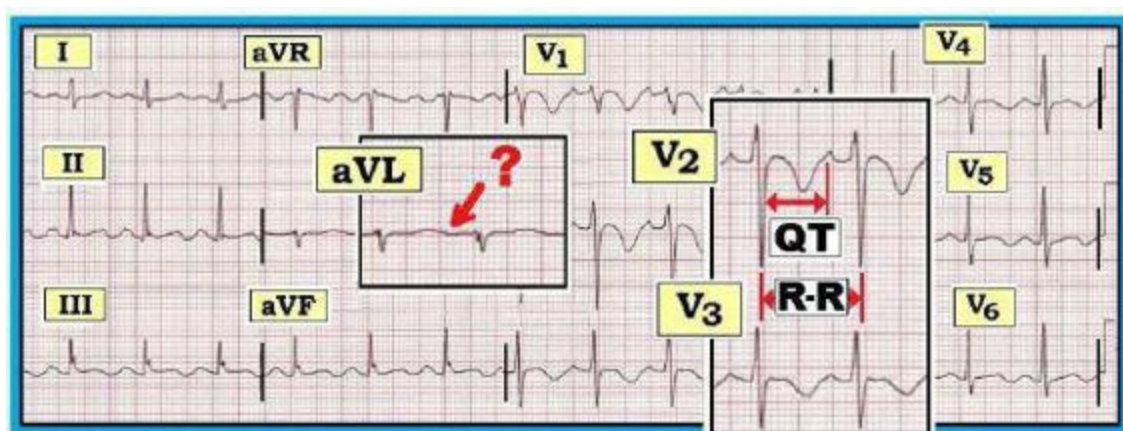


Figure 06.8-1: Is the QT interval prolonged? If so — What is the likely cause(s)?

Answer to Figure 06.8-1: The rhythm is sinus tachycardia at a rate just over 100/minute. The PR interval and QRS duration are both normal. However — the **QT interval** is **long**:

- As emphasized in Section 06.1 — the QT interval should be measured in a lead where you can clearly see the *end* of the T wave and in which the QT appears to be longest. As a result — a lead such as **aVL** should *not* be used, since the end of the T wave is *not* well defined in this lead (*red question mark in Fig. 06.8-1*).
- On the other hand — it is much easier to determine the limits of the T wave in *other* leads. This is especially true for **lead V2** — in which the QT interval is obviously *more* than half the R-R interval. **NOTE:** Although the “eyeball method” for assessing QT interval duration is admittedly *less* accurate when heart rate exceeds 90-100/minute (*Section 06.2*) — QT duration in Figure 06.8-1 is *so much more* than half the R-R interval, that the QT is undoubtedly prolonged *despite* the fact that the heart rate is relatively rapid.
- Regarding assessment of the *rest* of the ECG in Figure 06.8-1 — the most remarkable finding is **deep, symmetric T wave inversion** in most precordial leads.

IMPRESSION: Sinus tachycardia — *marked* QT prolongation — and diffuse *symmetric* T wave inversion. In addition to ischemia (*which is suggested by this symmetric T wave inversion*) — Think **“Drugs/Lytes/CNS”** as the categories of entities to consider in your differential diagnosis for the **markedly prolonged QT interval** seen here (**LIST #3** in Figure 06.2-1). Clinical correlation is *needed* to comment further on the most likely causes.

- Beyond-the-Core: The reason we say the QT interval is *markedly* prolonged — is that the QT literally makes up *more* than 2/3 of the R-R interval (See insert of leads V2, V3 in Figure 06.8-1). Thus *despite* the relatively rapid rate and the possibility of ischemia — strong consideration should be given to *drug-induced* QT prolongation — *severe* hypokalemia/hypomagnesemia — *and/or* CNS catastrophe as *contributing* causes (List #3).

06.9 – FIGURE 06.9-1: *Is the QT Long?*

We conclude this segment on assessment of the QT interval with one more 12-lead tracing (Figure 06.9-1). The rhythm is sinus tachycardia at a rate of ~150/minute. There is diffuse ST segment depression. Comment on the QT interval.

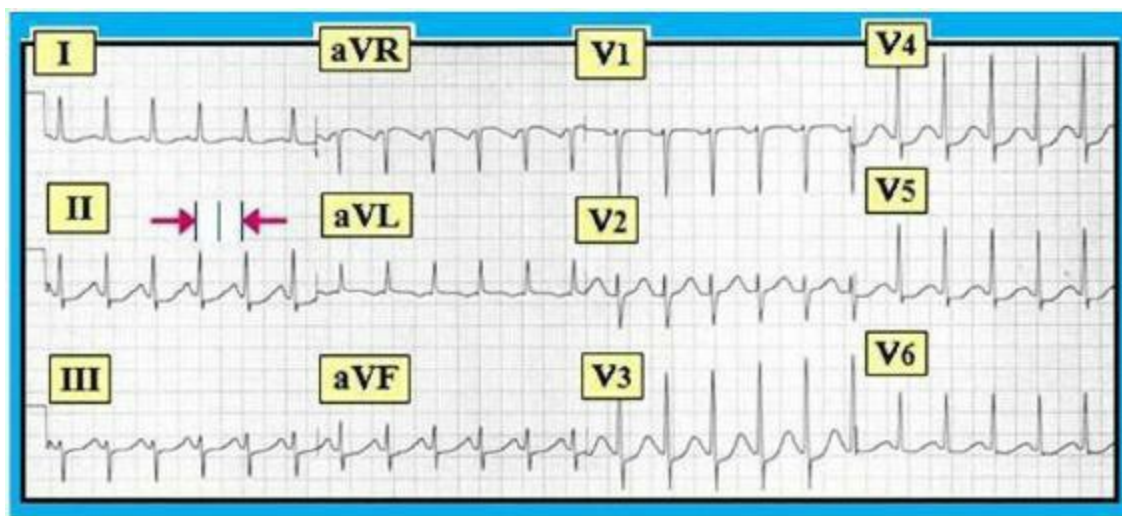
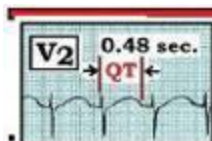


Figure 06.9-1: Sinus tachycardia at ~150/minute. There is diffuse ST depression. Comment on the QT interval (See text).

Answer to Figure 06.9-1: As stated — there is sinus tachycardia and diffuse ST segment depression. Clearly — a heart rate of ~150/minute is *too fast* to accurately apply the “eyeball method” for QT assessment. That said — a look at *multiple* leads in Figure 06.9-1 — suggests that the QT interval makes up as much as ~80% (*if not more*) of the R-R interval.

- While *numerical* determination of a QTc is not realistic for the ECG in Figure 06.9-1 (*because the rate is too fast*) — we strongly suspect the QT is significantly prolonged. Clinically — We again consider “*Drugs/Lytes/CNS*” as possible contributing causes.



Calculating the QTc

Beyond-the-Core: As emphasized in Section 06.5 — the *computerized* interpretation will automatically generate a QTc value for you that takes into account the patient's baseline heart rate. The ***computer-calculated QTc*** is usually quite accurate. As a result — a perfectly appropriate approach is to first *estimate* QT interval duration yourself by the “eyeball method” (*the QT should not be more than half the R-R interval*) — and, to then check this out with the QTc value provided by the computer:

- The **upper normal limit** for the **QTc** is **~450msec**. QTc values greater than this suggest QT prolongation.
- **Clinically** — concern about QT prolongation increases once the QTc *exceeds* 480 msec (*especially if >500 msec*)!

An advantage of incorporating the *computer-calculated* QTc value — is that doing so facilitates **serial comparison** of **QT duration** in a given patient. For example — *slight* prolongation of the QTc from a baseline of 430 msec to 445 msec in a patient started on Sotalol (*an antiarrhythmic agent known to significantly increase QT duration when used in higher doses*) would not necessarily mandate stopping the drug or even lowering the dose. However, continued *progressive* QT prolongation (*say, from 430 msec — to 445 msec — to 470 msec*) — would clearly require reassessment of the treatment regimen.

06.11 – BEYOND-the-Core: *Estimating the QTc Yourself*

For those providers with a desire to quickly estimate the QTc on their own without need to use the *computer-calculated* value — We propose the following:

- The formula for determining **QTc** = the QT that you measure — divided by the **square root** of the **R-R interval**. The “good news” is that at a heart rate of 60/minute — the R-R interval = 5 large boxes (= 1.0 second) — and the square root of one is one! This means that at a rate of 60/minute — the QTc is the QT interval that you measure.
- At heart rates *faster* than 60/minute — the QTc will be *more* than the QT that you measure (*because QTc varies inversely with the square root of the R-R interval — and the R-R interval for rates faster than 60/minute is less than one*).
- Determining the *square root* for *any* R-R interval *other than* an R-R interval of exactly 1.0 second is complex. *This is why a computer is needed for precise QTc calculation.* That said — We have devised a **simple correction factor** that can be used to rapidly *approximate* QTc duration.

Use of Our Proposed Correction Factor for QTc Estimation:

Determine the heart rate. Measure the QT interval in a lead where you can *clearly* see the end of the T

wave and in which the QT interval appears to be *longest*. Then **multiply** the QT interval you measure by the **correction factor** corresponding to your estimation of heart rate:

- Multiply by **1.0** for a heart rate of **~60/minute**.
- Multiply by **1.1** for a heart rate of **~75/minute**.
- Multiply by **1.2** for a heart rate of **~85/minute**.
- Multiply by **1.3** for a heart rate of **~100/minute**.

Applying the QTc Correction Factor:

Let's assume that the QT interval you *measure* is **0.40 second** (= *precisely 2 large boxes in duration*). IF the heart rate was **~60/minute** — then the QTc = the QT you measure = 0.40 second.

- IF on the other hand, the heart rate was **75/minute** — then we can estimate the QTc by multiplying the QT we measure (= *0.40 second*) by the **correction factor** of **1.1** = **~0.44 second**.
- IF instead the heart rate was **~85/minute** — then the QTc would be $0.40 \times 1.2 = \text{~0.48 second}$.
- IF instead the heart rate was **~100/minute** — then the QTc would be $0.40 \times 1.3 = \text{~0.52 second}$.
- Accuracy for QTc estimation decreases with rates substantially faster than 100/minute — so we do not offer additional correction factors beyond this rate.
- Practically speaking — there is no need to correct the QT for rate when there is bradycardia (*since change in the QTc is relatively small at rates below 60/minute*).

06.12 – FIGURE 06.12-1: Approximate the QTc

The 12-lead ECG in Figure 06.12-1 shows normal sinus rhythm at a rate just over 75/minute (*since the R-R interval is just under 4 large boxes in duration*).

- Assess the QT by the “eyeball method”. Is the QT interval likely to be normal or prolonged?
- Estimate the QTc by applying the appropriate *correction factor* from Section 06.11 that corresponds to a heart rate just over 75/minute.

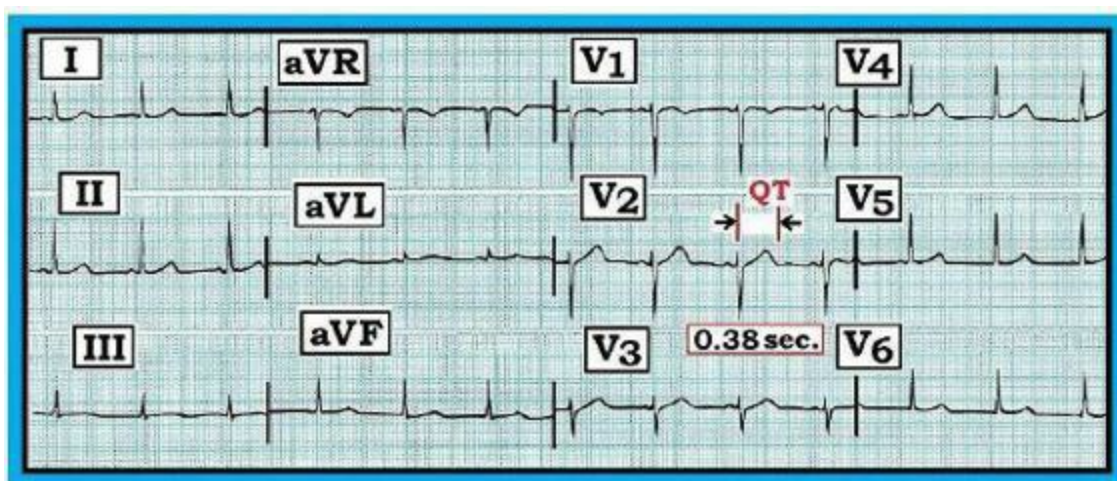


Figure 06.12-1: Sinus rhythm at a rate just over 75/minute. Assess the QT interval. Approximate the

QTc (See text).

Answer to Figure 06.12-1: As stated — there is sinus rhythm at a rate just over 75/minute.

- We select **lead V2** to measure the QT — because we can clearly see the end of the T wave in this lead. Alternatively We could have measured the QT in a number of other leads (ie, *leads II, V3, V4, V5*).
- By the “**eyeball method**” — the **QT appears** to be **normal** (*since the QT is not more than half the R-R interval*).
- As shown in Figure 06.12-1 — We **measure** the QT to be **~0.38 second** (*just a bit less than 2 large boxes in duration*).
- Given the heart rate *close to 75/minute* — We use a **correction factor of 1.1**. Multiplying 0.38 X 1.1 is *approximately 0.42 second* = the **QTc**. Since **420 msec** is clearly *less* than normal upper limit of 450 msec (*Section 06.10*) — we know that the **QTc** is **normal** even *without* referring to the *computer-calculated* value.

06.13 – FIGURE 06.13-1: *Approximate the QTc*

The 12-lead ECG in Figure 06.13-1 shows sinus tachycardia at a rate of ~100/minute (*since the R-R interval is ~3 large boxes in duration*). The QRS complex is narrow.

- Assess the QT by the “eyeball method”. Is the QT interval likely to be normal or prolonged?
- Estimate the QTc by applying the appropriate *correction* factor from Section 06.11 that corresponds to a heart rate of ~100/minute.

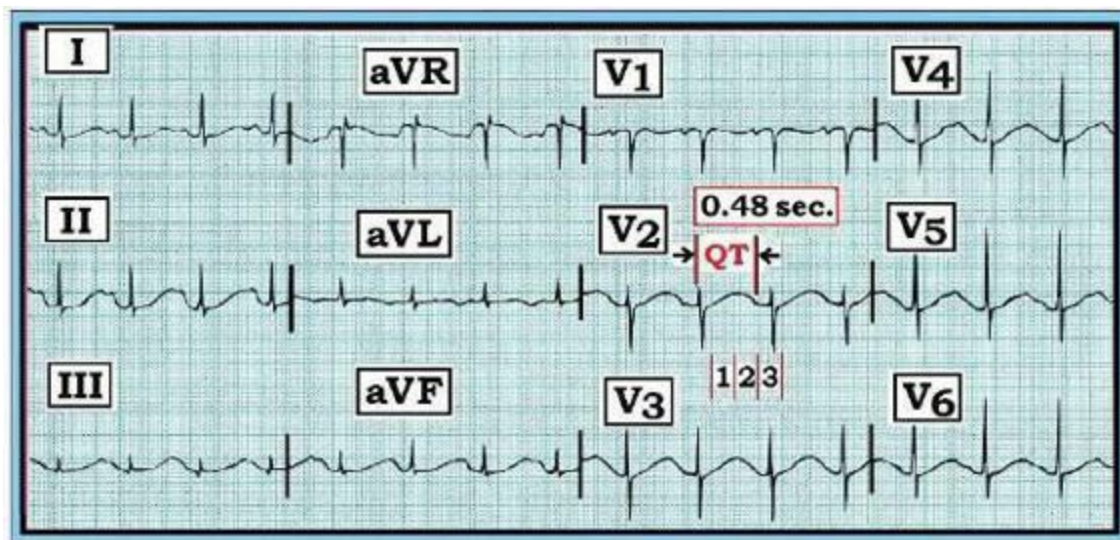


Figure 06.13-1: Sinus tachycardia at ~100/minute. Assess the QT interval. Approximate the QTc (See text).

Answer to Figure 06.13-1: As stated — there is sinus tachycardia at a rate of ~100/minute. The QRS complex is *narrow* — so any QT widening that may be seen is *not* the result of bundle branch block or other conduction defect.

- We again select **lead V2** to assess the QT. By the “*eyeball method*” — the **QT appears** to be **markedly prolonged**. Although heart rate is *faster* than is optimal for QT assessment — the QT takes up *more* than 2/3 of the R-R interval. There is therefore *no doubt* that the QT is prolonged.
- As shown in Figure 06.13-1 — We **measure** the **QT** to be **~0.48 second** (*easily more than 2 large boxes in duration*).
- Given the heart rate = **100/minute** — We use a **correction factor** of **1.3**. Multiplying 0.48 X 1.3 comes to a **QTc** of **over 600 msec**! Therefore — we know that the **QTc** is **dangerously prolonged** *without* having to refer to the *computer-calculated* value.

BOTTOM Line: This QTc Addendum (*Sections 06.10-thru-06.13*) is written solely for those wanting additional insight into clinical use of more precise QTc determination, including a *user-friendly* method for rapid estimation of the QTc on your own *without* need to refer to the *computer-calculated* value. We emphasize that this is advanced material.

- Most of the time — it suffices to simply use the “*eyeball method*” covered in Sections 06.1 and 06.2 for QT assessment.
- Awareness that *computerized* interpretations *automatically* provided you with a fairly accurate QTc value corrected for heart rate helps take QT interval assessment one step further. The normal upper limit for QT duration is ~450 msec. QTc values greater than this amount indicate QT prolongation. Clinical concern about QT prolongation increases with QTc intervals that *exceed* 480-500 msec. *Serial* QTc assessment may provide additional insight when *progressive* increase in QT duration is subtle.
- For those who want more — *Try out* our proposed method for rapid QTc estimation in Section 06.11.

Determining Axis

07.1 – Overview: *Limb Lead Location*

The mean **QRS Axis** may be defined as the average direction of the heart's electrical activity. A standard ECG is recorded by viewing the heart's electrical activity from 12 leads. Each lead records the heart's electrical potential from its own particular vantage point (**Figure 07.1-1**).

- As discussed in detail in Sections 03.1-thru-03.5 — the three **standard limb leads** are I, II, and III. Because electrical derivation of these leads is based on the premise of Einthoven's *equilateral* triangle — *each* of these leads is separated from each other by 60° , starting with **lead I** (at 0°) — followed by **lead II** (at $+60^\circ$) and **lead III** (60° further away at $+120^\circ$).
- The **augmented limb leads** are each separated from each other by 120° — and as such form a "Mercedes-Benz" triangle (dotted lines in **Figure 07.1-1**) — beginning with vertical **lead aVF** (at $+90^\circ$) — lateral **lead aVL** (at -30°) — and distant **lead aVR** (which we can usually ignore in axis determination — and need not recall its degree location).

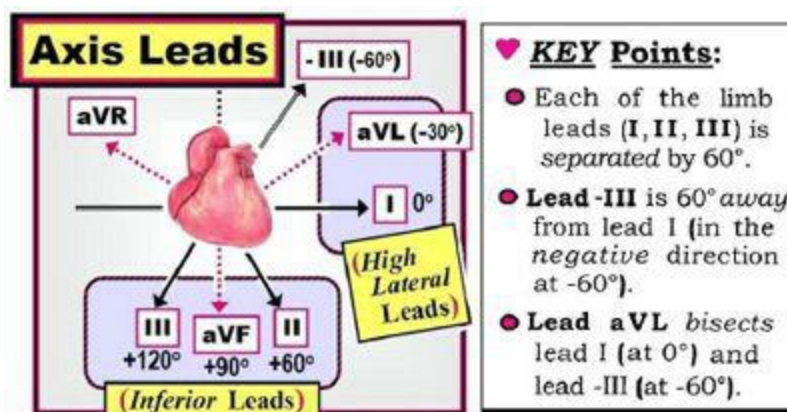


Figure 07.1-1: Limb lead location for calculation of axis.

Clinical Reality: Although axis determination is essential for diagnosis of the hemiblocks (*Section 07.9*) and may contribute to diagnosing RVH; pulmonary embolus; dextrocardia; and lead misplacement — most of the time, calculation of axis provides only *limited* clinical information.

07.2 – AXIS: *The Quadrant Approach*

Mean QRS axis is calculated in the *frontal* plane. The 2 **KEY** leads that are used for axis determination are **lead I** and **lead aVF**:

- Think of **Lead I** — as the "starting" point. As such, it is easy to remember that this *horizontal* lead is oriented toward 0° (**Figure 07.2-1**).
- Lead aVF — is oriented *perpendicular* to lead I (ie, *looking straight up from the Feet*). **Lead aVF** is therefore 90° away from lead I (or at $+90^\circ$).

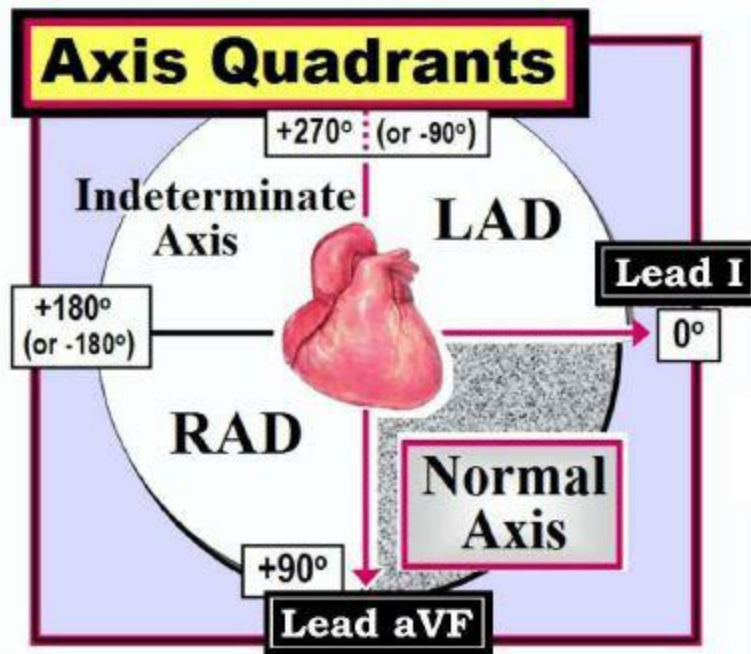


Figure 07.2-1: Axis quadrants. A **normal axis** is defined by the borders of leads I and aVF (ie, a normal axis lies between 0-to- $+90^\circ$).

KEY Point: It is easiest to define axis by **Quadrants**:

- A **normal axis** (See Figure 07.2-1) — is defined as lying *within* the limits of lead I (at 0°) and lead aVF (at $+90^\circ$).
- **LAD (Left Axis Deviation)** — lies between -1° to -90° .
- **RAD (Right Axis Deviation)** — lies between $+91^\circ$ to $+180^\circ$.
- An **indeterminate axis** lies between $+180^\circ$ and $+270^\circ$ (or *between -90° and -180°*). This quadrant is the *furthest* away from the heart — and is often referred to as the “northwest quadrant” — or — “No-man’s land”.

Clinical Note: Although by the **Quadrant Approach** we describe an axis of $+95^\circ$ as “RAD” and an axis of -10° as “LAD” — we emphasize that this *minimal* amount of axis deviation is not of clinical consequence. It is *rarely* important to be more precise than within 20-to- 30° of the actual axis.

07.3 – AXIS: The Concept of Net QRS Deflection

Determination of the mean axis **quadrant** can be made *at a glance* — by inspection (*and comparison*) of the **net QRS deflection** in lead I vs lead aVF. To determine the “net” QRS deflection in any given lead — *Mentally* subtract *negative* deflections from *positive* ones (Figure 07.3-1). It is “net area” that counts. For example:

- **Panel A** (in Figure 07.3-1) — is *all* positive.
- **Panel B** — is *predominantly* positive (*compared to the R wave, the s wave is small*).
- **Panel C** — is *predominantly* positive (*with a small q wave and a small s wave — but a tall R wave*).

- **Panel D** — is positive (*albeit with no more than a small R wave*).

NOTE: The reason we use both *small-case* and *large-case* letters in describing Q, R and S wave deflections — is to convey *relative size*. While we are *not* aware of any official distinction between *small-* vs *large-case* — We favor use of **3 mm** (*3 little boxes*) as our defining characteristic:

- Q, R, and S waves all manifest an amplitude of *at least 3 mm*.
- The amplitude of **q**, **r** and **s** waves is 3 mm or less. We generally use a *small-case* designation for 3 mm deflections that are narrow vs *large-case* designation for a 3 mm deflection that is wider. Thus, we describe the complex in **Panel C** of Fig. 07.3-1 as a **qRs** deflection.

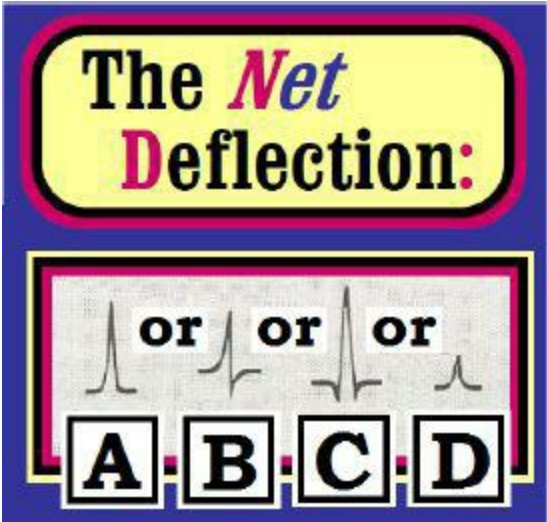


Figure 07.3-1: Illustration of how to assess “*net*” QRS deflection. Each of the examples shown here manifest a net *positive* deflection (*See text*).

07.4 – FIGURE 07.4-1: *How to Rapidly Determine Axis Quadrant*

We can determine the quadrant in which the axis lies *within seconds* by 2 simple steps: **Step #1:** Assess the *net* QRS deflection in leads I and aVF; and **Step #2:** Use the Table in **Figure 07.4-1**:

Axis Determination		
	Net QRS Deflection	
	Lead I	Lead aVF
Normal Axis	Positive	Positive
RAD	Negative	Positive
LAD	Positive	Negative
Indeterminate	Negative	Negative

Figure 07.4-1: Rapid determination of the axis quadrant based on the *net* QRS deflection in leads I and aVF. For example — IF *net* QRS deflection is positive in *both* leads I and aVF — then the axis is normal (*between 0-to-90°*).

07.5 – AXIS: Refining the Quadrant Approach

Using the Table in Figure 07.4-1 allows *near* instant determination of the axis quadrant. We can refine our estimate for axis by considering the following:

- IF the *net QRS deflection* in **lead I** (at 0°) is about the same as that for **lead aVF** (at +90°) — then the axis should lie *midway* between these leads (= *close to +45°*).
- Instead — IF the *net QRS deflection* in lead I is positive but clearly *exceeds* the net deflection in lead aVF — then the mean QRS axis lies *closer* to lead I (ie, *between 0° and +40°*).
- In contrast — the axis lies *closer* to lead aVF (*between +50° and +90°*) — IF the *net QRS deflection* in lead aVF is greater than it is in lead I.
- The axis is **perpendicular** to (ie, **90° away from**) a lead where the QRS complex is **isoelectric** (*equal parts positive and negative to the QRS*).
- **All you are doing is approximating.** Axis calculation need not be exact — *as long as your estimate is within 20-to-30° of the actual axis*. As a result — we often provide an **axis range** for our answer (ie, *The axis lies between +40° to +50°*).
- Realize that you can **further refine** your estimate of axis by looking at net QRS deflection in *other* limb leads. That said — this is usually not clinically needed, as will soon be apparent in review of the axis examples that follow.
- Final Note: The **computerized interpretation** is usually quite accurate for determination of heart rate, intervals and axis. IF you prefer — you can almost always depend on the axis calculated by the computer.

07.6 – FIGURE 07.6-1: What is the Axis?

Based on the *net* QRS deflection in leads I and aVF — Estimate the *mean* QRS axis in **Panels A** and **B** of Figure 07.6-1.

- In *which* of the 4 Quadrants does the axis lie? (*Feel free to refer back to Sections 07.4 and 07.5 in formulating your answer*).

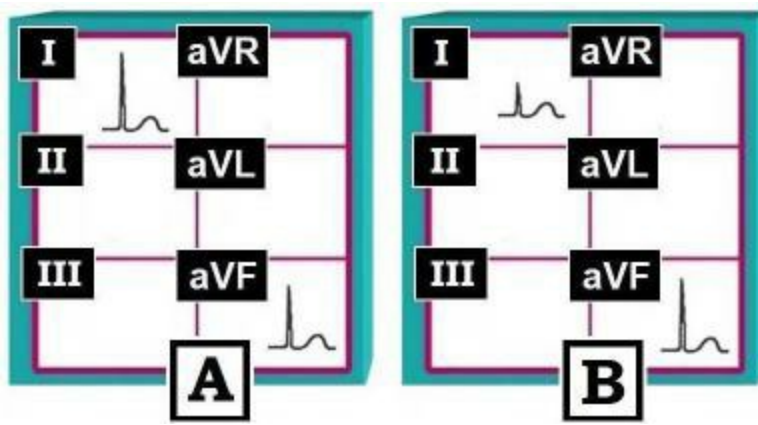


Figure 07.6-1: Estimate the axis for **Panels A** and **B**. In *which* of the 4 quadrants does the axis lie?

Answer to Figure 07.6-1: We can tell *at a glance* that the axis is normal (*between 0-to-90°*) in *both* **A** and **B** — because the *net* QRS deflection in *both* leads **I** and **aVF** is *positive* in *each* case. Use of a **quadrant diagram** allows us to refine our estimate for axis (**Figure 07.6-2**):

- **Panel A** (in **Figure 07.6-1**) — We *estimate* the axis at *between +30-40°*. The net deflection for *both* **I** and **aVF** is positive. The R wave in lead **I** is clearly *taller* than it is in lead **aVF**. As a result — the axis must lie *closer* to lead **I** (or *between 0-to-45°*). We illustrate this in **Panel A** of **Figure 07.6-2**.
- **Panel B** (in **Figure 07.6-1**) — We *estimate* the axis at *between +60-75°*. The net QRS deflection is decidedly *more* positive in **aVF** (**Panel B** in **Fig. 07.6-2**).

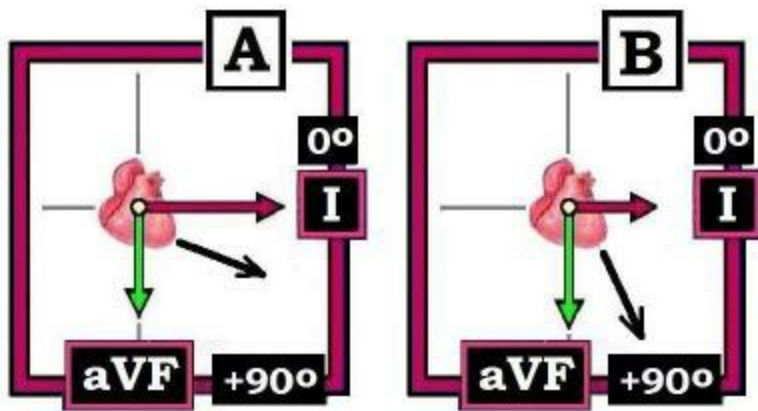


Figure 07.6-2: Quadrant diagram illustrating axis location (*black arrows*) for **Panels A** and **B** from **Fig. 07.6-1**.

07.7 – FIGURE 07.7-1: *What is the Axis?*

Estimate the *mean* QRS axis in **Panels C** and **D** of **Figure 07.7-1**:

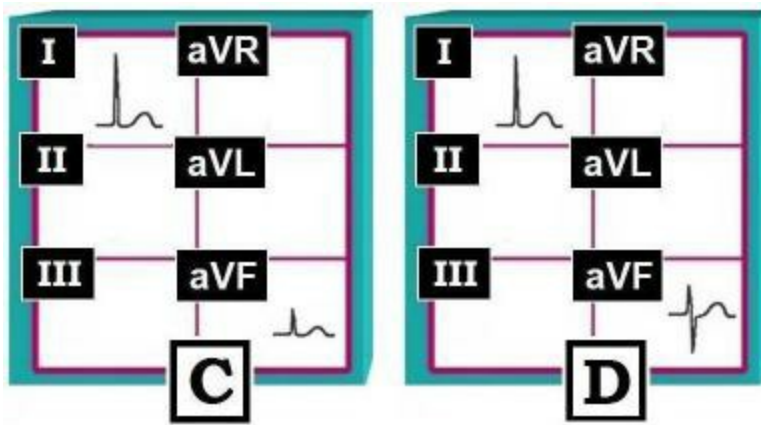


Figure 07.7-1: Estimate the axis for **Panels C** and **D**. In *which* of the 4 quadrants does the axis lie?

Answer to Figure 07.7-1: We can tell *at a glance* that the axis is *normal* for **C** (*positive deflection in I and aVF*) — but *not* normal in **D** (*negative deflection in one of these leads*). Use of a **quadrant diagram** allows a closer look (**Figure 07.7-2**):

- **Panel C** (in **Figure 07.7-1**) — We *estimate* the axis at *between* $+10\text{-}20^\circ$. Relative positivity of the *net* deflection in lead I compared to aVF is even *more* marked than it was for **Panel A** in **Figure 07.6-1**. The axis in **Panel C** must therefore lie *very close* to lead I (*as shown in Panel C of the quadrant diagram in Figure 07.7-2*).
- **Panel D** (in **Figure 07.7-1**) — There is at least **slight LAD** (**Left Axis Deviation**) — since *net* QRS deflection is *negative* in lead aVF. As we will see momentarily in discussion of hemiblocks (**Section 07.9**) — use of lead II will be needed to determine if the amount of LAD is more or less than -30° . We illustrate slight **leftward axis** for **Panel D** in the corresponding *quadrant diagram* shown in **Figure 07.7-2**.

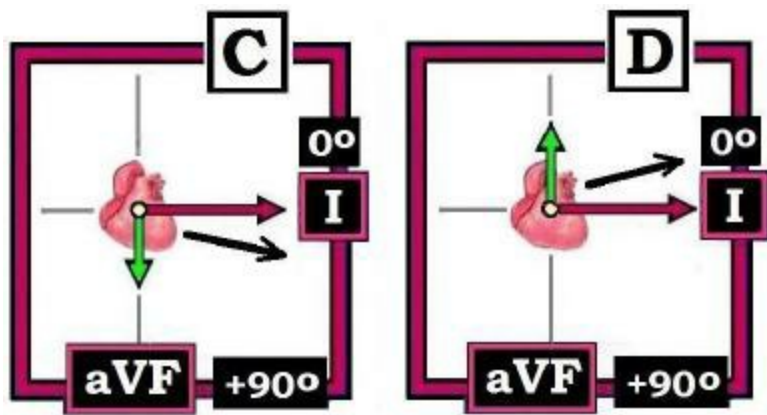


Figure 07.7-2: Quadrant diagram illustrating axis location (*black arrows*) for **Panels C** and **D** from **Fig. 07.7-1**.

07.8 – FIGURE 07.8-1: What is the Axis?

Estimate the *mean* QRS axis in **Panels E** and **F** of **Figure 07.8-1**:

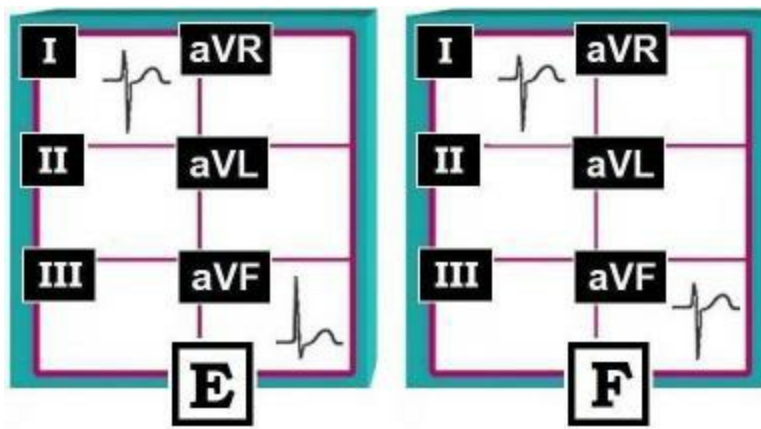


Figure 07.8-1: Estimate the axis for **Panels E** and **F**. In *which* of the 4 quadrants does the axis lie?

Answer to Figure 07.8-1: We can tell *at a glance* that the axis is not normal for **E** or **F** — because *net* QRS deflection is *not* positive in *both* leads **I** and **aVF**. Use of a **quadrant diagram** allows a closer look (**Figure 07.8-2**):

- **Panel E** (in **Figure 07.8-1**) — There is **RAD** (*Right Axis Deviation*) — since *net* QRS amplitude in lead **I** is clearly negative (*the S in lead I is deeper than the R wave is tall*). We estimate mean QRS axis to be *between* $+100^\circ$ -to- $+110^\circ$. We illustrate this in **Panel E** of the **Figure 07.8-2 quadrant diagram**. **Clinical NOTE:** It is sometimes difficult to accurately determine the specific number of degrees with right axis deviation. That said — it is not overly important whether the axis is $+105^\circ$, or for that matter $+120^\circ$. What counts clinically — is that there is *definite* RAD.
- **Panel F** (in **Figure 07.8-1**) — The **axis** is **indeterminate** — since the *net* QRS deflection is *negative* in *both* leads **I** and **aVF**. **Clinically** — *it no longer matters* how many degrees the axis is. All that counts is that axis location is in the upper right (*indeterminate*) quadrant (**Panel F** in the **Figure 07.8-2 quadrant diagram**). **PEARL:** — The most common clinical conditions associated with an *indeterminate* axis are: **i)** RVH; **ii)** COPD; **and iii)** Large body habitus.

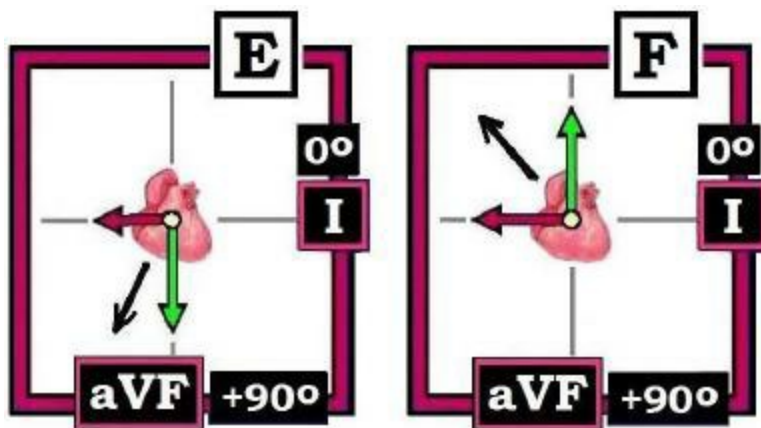


Figure 07.8-2: Quadrant diagram illustrating axis location (*black arrows*) for **Panels E** and **F** from **Fig. 07.8-1**.



07.10 – Hemiblocks: *Anatomic Considerations*

As seen below in **Figure 07.10-1** — after the electrical impulse arrives at the **AV Node** — it travels down the **Bundle of HIS**. From there, the ventricular conduction system divides into the slender **Right Bundle Branch** — and — the much thicker **Common Left Bundle Branch**.

- The *Common Left Bundle Branch* divides into 2 parts: the ***Anterior*** and ***Posterior Hemifascicles***. Development of a “***hemiblock***” — simply entails a defect in conduction over one of these hemifascicles.
- Note in **Figure 07.10-1** — that the ***posterior hemifascicle*** is anatomically much *thicker* than the ***anterior hemifascicle***. This is one reason why LPHB is uncommon. The other reason is a richer (*dual*) blood supply compared to the anterior hemifascicle. As a result — it is ***rare*** to see **LPHB** as an ***isolated defect*** in a patient *without* significant heart disease. Most patients with LPHB have *bifascicular block (RBBB/LPHB — See Sections 07.20, 07.21)*.

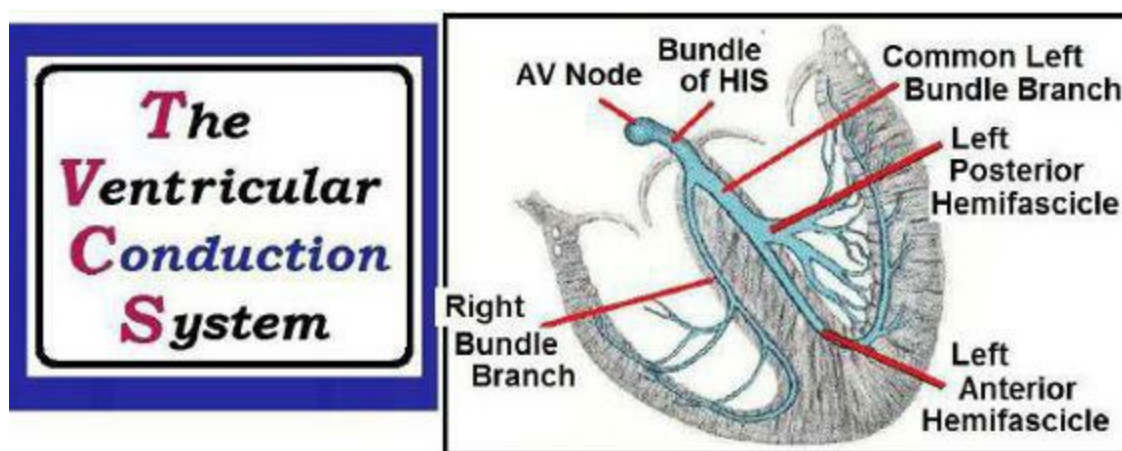


Figure 07.10-1: The ventricular conduction system. Note how much *thicker* the left *posterior* hemifascicle is compared to the anterior hemifascicle and the right bundle branch.

KEY Clinical Point: The *anatomic* diagram in **Figure 07.10-1** is simplified. In reality — there are *millions* of fibers in the ventricular conduction system with many potential anatomic variants on the arrangement shown. That said, *most* of the time — conduction fibers to the left ventricle are arranged in two large groups (*hemifascicles*) — one of which is situated slightly in front (*anterior*) of the other.

- The benefit of accepting the arrangement in **Figure 07.10-1** as the general anatomic model, is that it greatly ***simplifies diagnosis*** of the **hemiblocks**!

07.11 – *Advanced Concept: LSFB (a 3rd type of Fascicular Block)*

Beyond-the-Core: In a significant percentage of patients — a **3rd (septal) hemifascicle** appears to exist (*not shown in Fig. 07.10-1*). The collection of fibers that make up this **septal hemifascicle** is thinner, more variable in distribution than are fibers in the anterior and posterior hemifascicles, and generally manifests a shorter refractory period. As its name implies — fibers from the **septal** hemifascicle contribute to early activation of a portion of the ventricular septum in certain patients.

- As a result — a 3rd type of *partial* LBBB conduction block (*in addition to LAHB and LPHB*) may occur = **LSFB (Left Septal Fascicular Block)**.
- **LSFB is not commonly recognized on ECG.** This is because of several reasons: **i)** the existence of this 3rd hemifascicle is *not* well appreciated; **ii)** LSFB is uncommon; and **iii)** ECG manifestations of LSFB when it *does* occur are often masked by *simultaneous* occurrence of *other* conduction defects. Practically speaking — Do NOT be concerned about recognizing LSFB unless you are a student of *advanced* arrhythmia interpretation.
- **BOTTOM Line:** Our reason for mentioning the existence in some patients of a **septal** hemifascicle and therefore a potential *3rd type* of fascicular block is twofold: **i)** to shed light on the otherwise unexplained *coming-and-going* of septal q waves (*or of the initial small r wave in V1, V2*) that is sometimes seen in some challenging arrhythmias (*may be due to intermittent LSFB aberrant conduction*); and **ii)** to emphasize that the **anatomic arrangement** presented in **Figure 07.10-1** is a **simplification**. This simplification works well for understanding the general anatomic pattern of the ventricular conduction system — and it allows *rapid* diagnosis of the hemiblocks (*Section 07.12*).

07.12 – Hemiblocks: *An Approach to Rapid ECG Diagnosis*

Agreement among experts is *lacking* regarding diagnostic criteria for hemiblock. Some experts specify a certain number of degrees in axis deviation — whereas others favor QRS morphology change rather than axis criteria. Fortunately — ECG diagnosis of hemiblocks can be simplified into an *equally* accurate yet **user-friendly approach** that allows classification in less than 5 seconds. This approach is based on the following concepts:

- There are **2 hemiblocks** that we need to concern ourselves with: **anterior** and **posterior**. Life is simpler (*and no less accurate in the overwhelming majority of cases*) if we do not initially concern ourselves about the infrequent, *difficult-to-diagnose* 3rd type of conduction defect = LSFB (*Section 07.11*).
- Of the 2 types of hemiblock that we do need to concern ourselves with — **Left Posterior HemiBlock (LPHB)** — is **rare!** As already emphasized (*Section 07.10*) — the reason LPHB is so uncommon is that the *posterior* hemifascicle is much *thicker* anatomically and it has a *dual* blood supply (*from the left and right coronary arteries*); the anterior hemifascicle does not.
- **Even experts** often **do not agree** on the ECG diagnosis of **LPHB**. As a result — You are probably *none the worse* if you *never* diagnose LPHB. On those uncommon occasions when LPHB *does* occur — it will almost always be seen in association with RBBB (*See Section 07.21 and 07.25*).

- In contrast — diagnosis of **LAHB** (**L**eft **A**nterior **H**emi-**B**lock) — is far more common (>98% of hemiblocks in our experience). ECG **diagnosis** of **LAHB** becomes easy — IF you accept as your criterion for diagnosis recognition of a **pathologic left axis** (Section 07.13).

07.13 – LAHB: ECG Diagnosis = “pathologic” LAD

Expert electrocardiographers do not agree on how to define LAHB. Some define it by the number of degrees (*be this requiring a leftward axis more negative than -30° , -45° , or -60°*). Others maintain that *rather than axis* — it is QRS morphology in the limb leads that defines LAHB. Life is “**simpler**” (and equally accurate) — IF you **equate** **pathologic LAD** = **LAHB**. Consider the following:

- Some LAD (ie, -10° to -20°) — is common and not necessarily abnormal.
- We define a **pathologic LAD** — as a left axis *more negative* than -30° . It is easy to tell IF **pathologic LAD** is present. *You only need to look at lead II* (Figure 07.13-1):

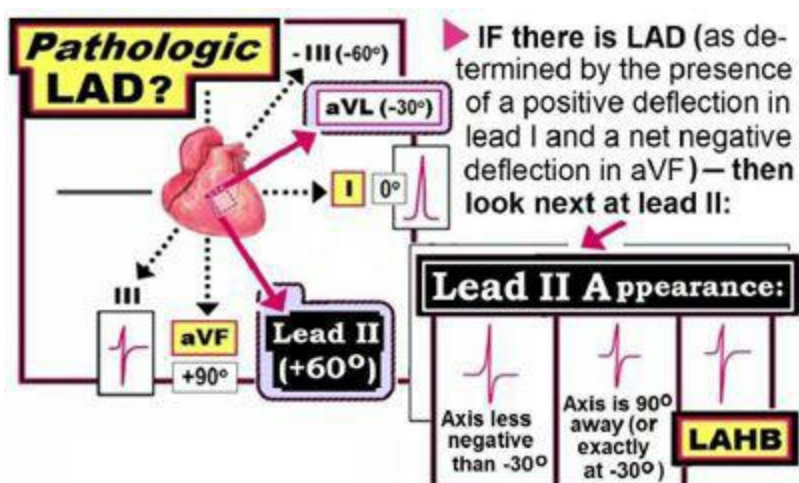


Figure 07.13-1: Diagnosis is simplified by **defining** **LAHB** as a “pathologic” left axis (= a net deflection in lead II that is more negative than positive).

KEY Summarizing Point: For practical purposes — We equate the ECG diagnosis of **LAHB** with the finding of **pathologic LAD** (which we define as a mean QRS axis more negative than -30°).

- Assuming lead I is positive (*as it almost always is*) — then the *amount* of LAD is “**pathologic**” — IF the **net deflection** in **Lead II** is **negative** (See *Lead II Appearance* in the lower right portion of Figure 07.13-1).

07.14 – FIGURE 07.14-1: Is there LAD? IF so — Is there LAHB?

Apply the quadrant approach to determine the axis for the 12-lead ECG shown in Figure 07.14-1:

- Is there LAD? If so — Is there also LAHB?
- Which lead should be used to determine if LAHB is or is not present in Figure 07.14-1?

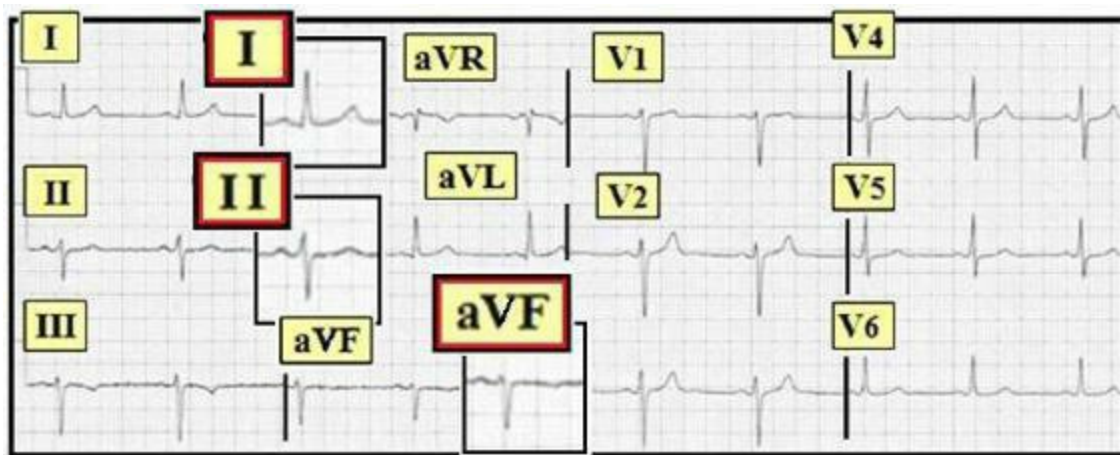


Figure 07.14-1: Estimate the axis. Is there LAD? If so — Is there also LAHB? (*See text*).

Answer to Figure 07.14-1: It is easy to determine the axis for the 12-lead ECG shown in Fig. 07.14-1 by use of the *quadrant* approach:

- The *net* QRS deflection in lead aVF is negative. It is positive in lead I.
- By the Table in [Figure 07.4-1](#) — **LAD** is therefore present. The situation is similar to the example illustrated by **Panel D** in Section 07.7, which we reproduce below in [Figure 07.14-2](#).
- Once established that there is LAD — We determine IF the amount of LAD is “*pathologic*” by looking at **lead II**.
- *Pathologic* LAD is clearly present in [Figure 07.14-1](#) — since the *net* QRS deflection in lead II is decidedly more negative than positive. Therefore — there is **LAHB** in [Figure 07.14-1](#). We estimate the mean QRS axis in this tracing to be *at least* -40° .

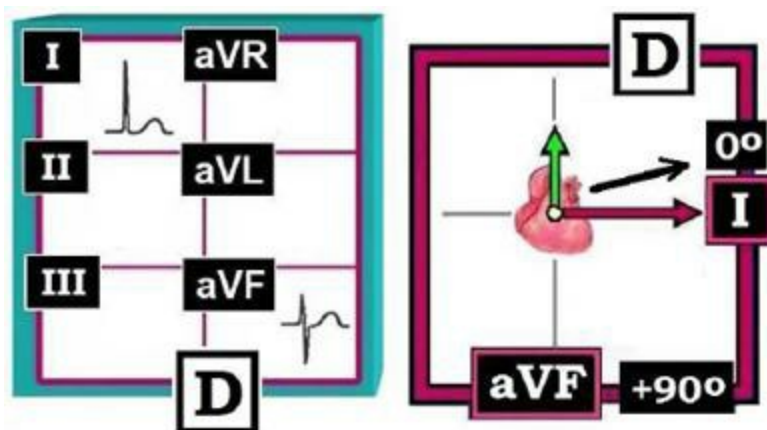


Figure 07.14-2: We reproduce Panel D from Section 07.7. The situation is similar to that shown in [Figure 07.14-1](#). There is **LAD** — because the *net* QRS deflection is positive in lead I and negative in lead aVF. We would need to see **lead II** in order to determine IF the amount of LAD was “*pathologic*” (ie, *more negative than* -40°).

07.15 – SUMMARY: *ECG Diagnosis of LAHB in 3 Seconds*

Panels **G** and **H** in [Figure 07.15-1](#) summarize how to tell *within* seconds IF there is LAHB:

- **Panel G** — By the quadrant approach, the *more-negative-than-positive* deflection in lead aVF defines there to be LAD. However the lead II deflection is not more negative than positive. Therefore the axis is not more negative than -30° in **Panel G**, which means there is **LAD but not LAHB**. In fact — the *isoelectric* appearance in lead II means that the axis is virtually perpendicular to (90° away from) lead II — or *precisely* at -30° .
- **Panel H** — The once again *more-negative-than-positive* deflection in lead aVF defines there to be LAD. This time the lead II deflection is more negative than positive. Therefore the LAD is *sufficiently leftward* to qualify as **LAHB**. We estimate the mean QRS axis to be *at least* -40° .

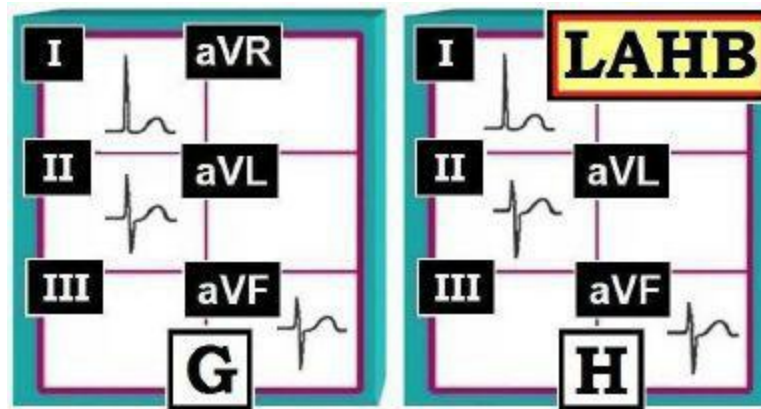


Figure 07.15-1: Panels G and H both show LAD. There is LAHB in **H** but not in G (because *net QRS deflection is negative in lead II for H, but not in G*).

07.16 – Bifascicular Block



07.17 – Definition/Types of Bifascicular Block

The term “**bifascicular**” block — implies that *more* than a single major branch of the ventricular conduction system is not functioning. The defect in conduction may be transient or permanent. Practically speaking — there are **2 Types** of **Bifascicular Block**:

- **RBBB/LAHB** = RBBB *plus* LAHB.
- **RBBB/LPHB** = RBBB *plus* LPHB.

Clinical Note: Semantically — **complete LBBB** is also a type of “**bifascicular**” block — since there is implication of failed conduction (*by definition*) in *both* anterior and posterior hemifascicles when there is LBBB. That said — We generally do not think of LBBB as “**bifascicular**” block. This leaves us with the 2 types of hemiblock cited above (*RBBB + either LAHB or LPHB*).

07.18 – RBBB/LAHB: ECG Recognition

By far — the most common form of **bifascicular block** is the *combination* of **RBBB/LAHB**. The *schematic* tracing in Figure 07.18-1 shows the typical ECG appearance of this conduction defect. Note the *underlying* rhythm is sinus (*upright P wave in lead II*) and the QRS looks to be wide. We focus attention on the 3 key leads for diagnosing BBB (*leads I, V1, V6* — *Section 05.2*) — *plus* on lead II.

- **RBBB** — is recognized by its characteristic appearance in the 3 key leads (*rSR' with taller right rabbit ear in V1; wide terminal S wave in leads I, V6*).
- That there *also* is **LAHB** — is seen from the **negative net deflection** in **lead II** (*Section 07.13*).

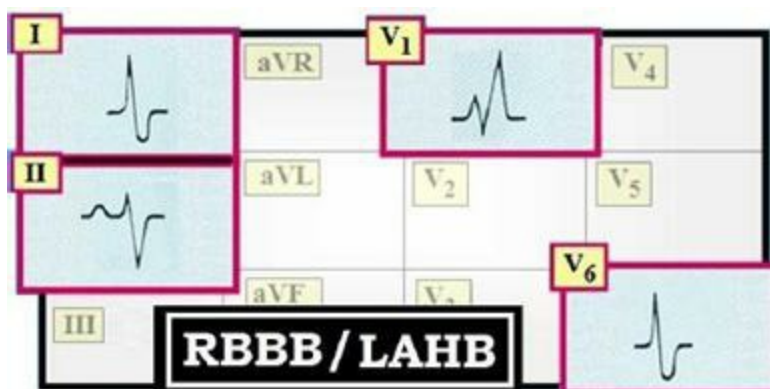


Figure 07.18-1: RBBB/LAHB. **RBBB** — is diagnosed by the rSR' in lead V1 *with* wide, terminal S waves in I, V6. **LAHB** — is diagnosed by the decidedly *more-negative-than-positive* deflection in lead II (*See text*).

07.19 – The Meaning of “Axis” when there is RBBB

The concept of “axis” in the setting of RBBB is problematic. Calculation of axis is based on the **average direction** of the heart’s **electrical activity** in the frontal plane. In the presence of **RBBB** — electrical activity first goes **left-to-right** (*as the septum is depolarized*) — then **right-to-left** (*as the left ventricle is depolarized*) — and then left-to-right again as the *blocked* right ventricle is finally activated via slow myocardial conduction through *nonspecialized* fibers. So in the setting of RBBB — the “average direction” of the heart’s electrical activity does not reflect the actual *zig-zag* path of the depolarization impulse.

- **Bottom Line:** — *Don’t worry about “axis” when RBBB is present!*
- All we care about when there is **RBBB** — is *which* of these **3 possible settings** is operative: **i) RBBB** and *nothing* else; **ii) RBBB plus LAHB**; or **iii) RBBB plus LPHB**.
- The ECG picture of **RBBB/LAHB** is *easy* to recognize. In addition to RBBB — We can tell at a glance IF **LAHB** is *also* present by a **look** at **lead II** to see IF the *net* QRS deflection is *predominantly* negative (Figure 07.18-1).

07.20 – Clinical Implications of Bifascicular Block

The clinical significance of virtually *any* conduction system defect depends on the setting in which it occurs. **Isolated RBBB** may sometimes occur in otherwise healthy individuals *without* underlying heart disease — in which case RBBB is *unlikely* to have any adverse prognostic implications (*Section 05.5*). In contrast — a **new finding** of **RBBB** in the setting of acute evolving MI implies *ongoing* conduction system damage (*suggesting a larger infarct size and possible need for a pacemaker*).

- **Bifascicular block** — clearly implies a more important conduction defect than *isolated* RBBB. That said — IF the patient is otherwise *asymptomatic*, then *bifascicular* block from **RBBB/LAHB** may *not* necessarily convey any adverse prognostic implications. *Combined* RBBB/LAHB is seen surprisingly often as an *incidental* finding in an otherwise asymptomatic older individual.
- On the other hand — IF *new* RBBB/LAHB develops in the setting of acute coronary syndrome — the extent of damage is probably large (*and the patient may soon need a pacemaker*). *Clinical* context is everything!
- The *other* form of *bifascicular* block = **RBBB/LPHB** — is **much less common**. As previously stated (*Section 07.10*) — the reason LPHB is so uncommon relates to its anatomy (*much thicker fascicle*) and *dual* blood supply. As a result, IF **RBBB/LPHB** *does* occur — it implies a more extensive conduction system defect (*with potentially more severe prognostic implications*).

07.21 – RBBB/LPHB: ECG Recognition

The *uncommon* form of **bifascicular block** — is the *combination* of **RBBB/LPHB**. The *schematic* tracing in Figure 07.21-1 shows the typical ECG appearance of this conduction defect. Note the *underlying* rhythm is sinus (*upright P wave in lead II*) and the QRS looks to be wide. We focus

attention on the 3 *key* leads for diagnosing BBB (*leads I, V1, V6* — *Section 05.2*) — plus on lead II.

- **RBBB** — is recognized by its characteristic appearance in the 3 *key* leads (*rSR' with taller right rabbit ear in V1; wide terminal S wave in leads I, V6*).
- That there *also* is **LPHB** — is seen from the presence in **lead I** of a **deep *straight* initial descent** of the **S wave** in this lead. In support of the diagnosis of RBBB/LPHB — is the presence of a qR pattern in lead II with a relatively tall R wave in this lead.
- **NOTE:** Leads II, III and aVF all typically manifest a *similar* ECG appearance when there is RBBB plus LPHB (ie, a qR pattern with relatively tall R wave). That said — We can *simplify* the process of recognizing RBBB/LPHB by focusing attention on QRS appearance in the 3 *key* leads (*I, V1, V6*) — plus on lead II — as is done in **Figure 07.21-1**.

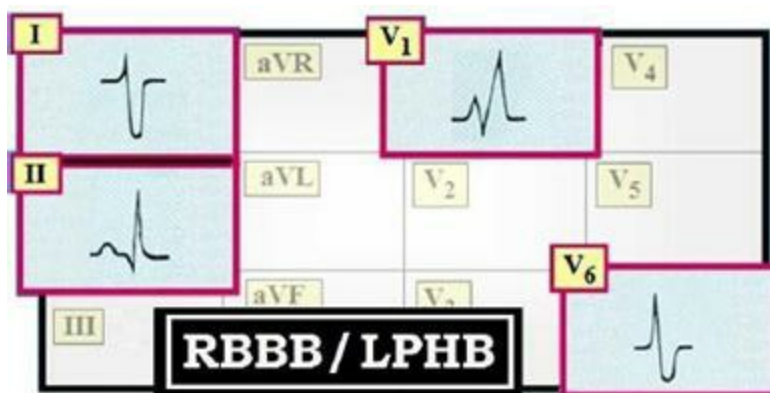


Figure 07.21-1: RBBB/LPHB. **RBBB** — is diagnosed by the rSR' in lead V1 with wide, terminal S waves in I, V6. **LPHB** — is diagnosed by the very *steep* initial descent of the S wave in lead I. In addition — Note the qR pattern in lead II with relatively tall R wave (*See text*).

07.22 – RBBB/LPHB: *Finer Points on ECG Recognition*

The experts often do not agree on the diagnosis of *bifascicular* block from **RBBB/LPHB**. The *KEY* to recognizing this form of bifascicular block is that once *complete* RBBB is identified — Focus attention on **S wave *descent* in lead I**.

- As emphasized in Section 05.4 — a wide *terminal* S wave in *lateral* leads I, V6 is part of the *expected* QRS morphology seen with *complete* RBBB (**Panel A** in Figure 07.22-1). That said — the initial (*straight portion*) of the S wave in lead I is *not* nearly as steep with *isolated* RBBB — as it is when LPHB is also present (*red arrow in Panel B of Fig. 07.22-1*).

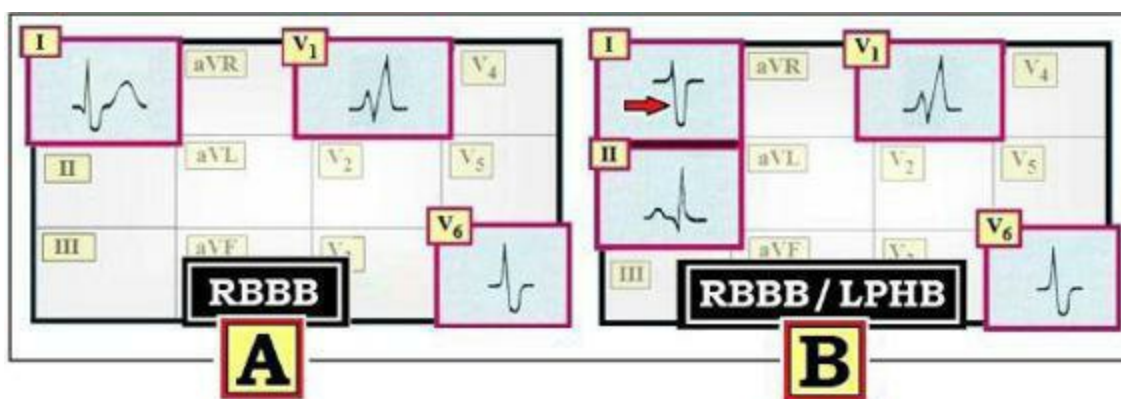


Figure 07.22-1: RBBB by itself (**Panel A**) — compared to *bifascicular* block when there is RBBB *plus* LPHB (**Panel B**). Note the very *steep* initial descent of the S wave (*red arrow*) in lead I when LPHB is also present (*See text*).

Beyond-the-Core: Because the *posterior hemifascicle* lies not only posterior, but also inferior and rightward (*in 3 dimensions*) with respect to the anterior hemifascicle — the *blocked* portion of the QRS deflection in lead I is directed inferior and to the right. This is the reason for the *steep* negative S wave descent in lead I and the predominant *positive* R wave in leads II, III, and aVF (**Panel B** in Fig. 07.22-1).

- Typically — there will also be a slender initial r wave in lead I and a small narrow q wave in the *inferior* leads (II, III, aVF) when there is **RBBB/LPHB**. This is because the *initial* direction of left ventricular depolarization with RBBB/LPHB is toward that part of the LV supplied by the *intact* left anterior hemifascicle. Given the *relative* leftward and superior orientation of the **left anterior hemifascicle** (*compared to the relative rightward and inferior orientation of the posterior hemifascicle*) — initial electrical activity with RBBB/LPHB tends to produce a small r in lead I and a small q in inferior leads (**Panel B** in Figure 07.22-1). That said — these subtle ECG features might be altered if in addition to RBBB/LPHB there has been prior scarring or infarction.
- Bottom Line:** — Remember that *bifascicular* block from **RBBB/LPHB** is uncommon in clinical practice. ECG diagnosis is often challenging and encompasses an *advanced* subject area about which cardiologists do *not* always agree. You are probably *none the worse* if you *never* diagnose this entity. Our goal is merely to highlight the ECG features we find helpful for recognizing RBBB/LPHB when it is present. Realize that once you do identify this form of *bifascicular* block — it is almost certain that your patient has *extensive* underlying heart disease.

07.23 – FIGURE 07.23-1: *Is there Bifascicular Block?*

Assess the ECG in Figure 07.23-1 for the presence of conduction defects.

- Is there *bifascicular* block?

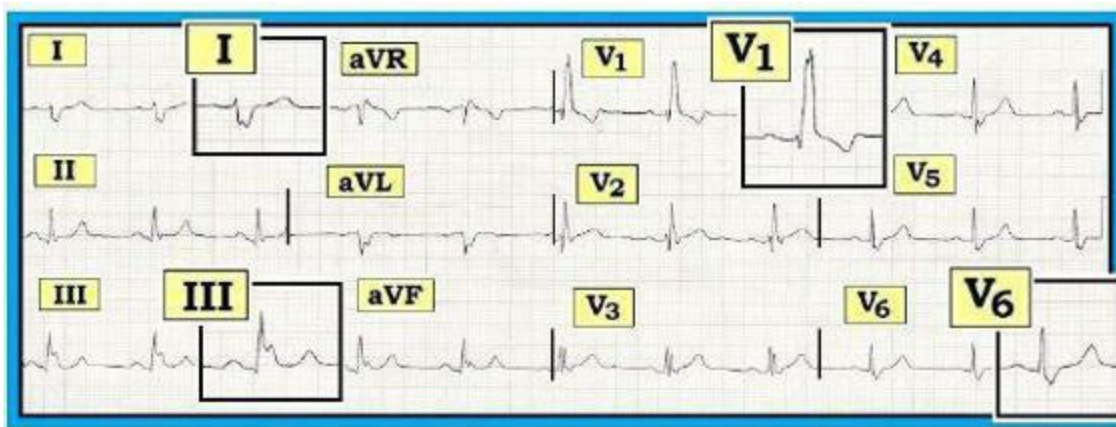


Figure 07.23-1: Sinus rhythm with **RBBB/LPHB** (See text).

Answer to Figure 07.23-1: The rhythm is sinus. The QRS complex is wide.

- **RBBB** — is recognized by its characteristic appearance in the 3 key leads (*rSR' with taller right rabbit ear in V1; wide terminal S wave in leads I, V6*).
- In addition, there is **LPHB** — as diagnosed by the presence in lead I of a **deep straight initial descent** of the S wave in this lead. In support of the diagnosis of RBBB/LPHB — is the presence of a qR pattern in leads II, III, aVF with a relatively tall R wave in these leads (*especially in lead III*). Thus, there is **bifascicular block (RBBB/LPHB)**.

07.24 – FIGURE 07.24-1: *Is there Bi- or Tri-Fascicular Block?*

Assess the ECG in Figure 07.24-1 for the presence of conduction defects.

- Is there *bifascicular* block?
- What is “*trifascicular*” block?

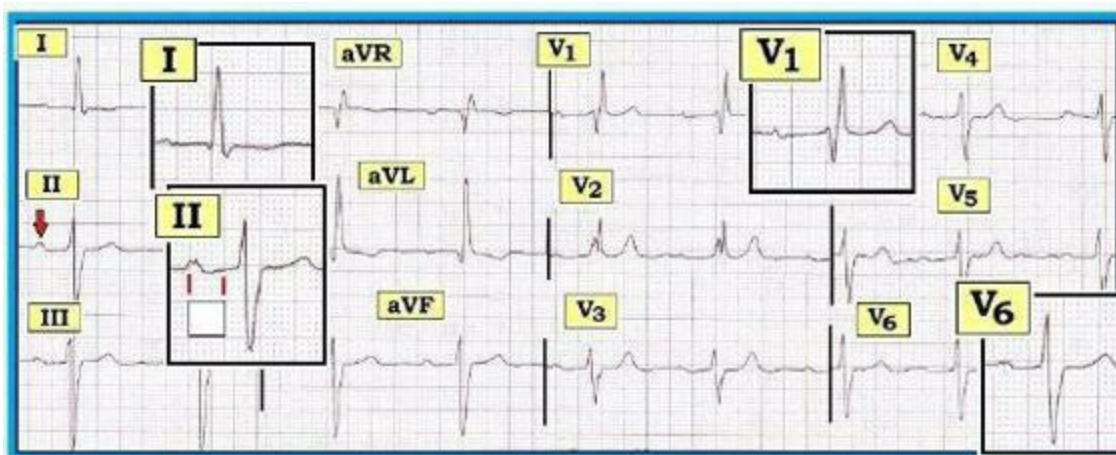


Figure 07.24-1: Sinus rhythm with **RBBB/LAHB**. Is there *trifascicular* block? (See text).

Answer to Figure 07.24-1: The rhythm is sinus (*upright P wave in lead II, as shown by the red arrow*). The PR interval is prolonged (*clearly more than a large box in duration*) — so there is **1st degree AV block** (Section 02.70).

- The QRS complex is wide. Blowup inserts from the 3 *key* leads (I, V1, V6) — confirm **RBBB** (*rSR'* in V1; wide terminal S waves in leads I and V6). NOTE: Even though the S wave in lead I is *not* deep — it is relatively wide, and this satisfies criteria for RBBB (Section 05.4).
- LPHB is clearly not present in Figure 07.24-1 — because the S in lead I is small *without* a steep descent.
- On the other hand — **LAHB** is present, because the QRS deflection in lead II is *predominantly* negative (Section 07.18). Therefore — there is **bifascicular block** (RBBB/LAHB).

Beyond-the-Core: The term, “**trifascicular**” block implies impaired conduction in *all* 3 of the major conduction fascicles: **i)** the right bundle branch; **ii)** the left *anterior* hemifascicle; and **iii)** the left *posterior* hemifascicle.

- Clinically — Diagnosis of *trifascicular* block is usually not possible from the surface ECG. We simply *cannot tell* IF PR interval prolongation in a patient with *bifascicular* block (as in Figure 07.24-1) is due to AV nodal disease or disease in the remaining conducting fascicle. Therefore — We would interpret this ECG as showing *bifascicular* block (RBBB/LAHB) plus 1st degree AV block.

07.25 – FIGURE 07.25-1: Isolated LPHB vs Right Axis Deviation?

We conclude our discussion on hemiblocks and *bifascicular* block with the 12-lead ECG shown in **Figure 07.25-1** — obtained from an otherwise healthy young adult male seen in the office for an insurance physical. The rhythm is sinus (*probable sinus arrhythmia*) — and the QRS complex does *not* appear to be prolonged.

- **RAD** (Right Axis Deviation) is clearly present — as determined by a predominantly *negative* QRS in lead I with an *upright* QRS in lead aVF.
- Does this patient have LPHB?

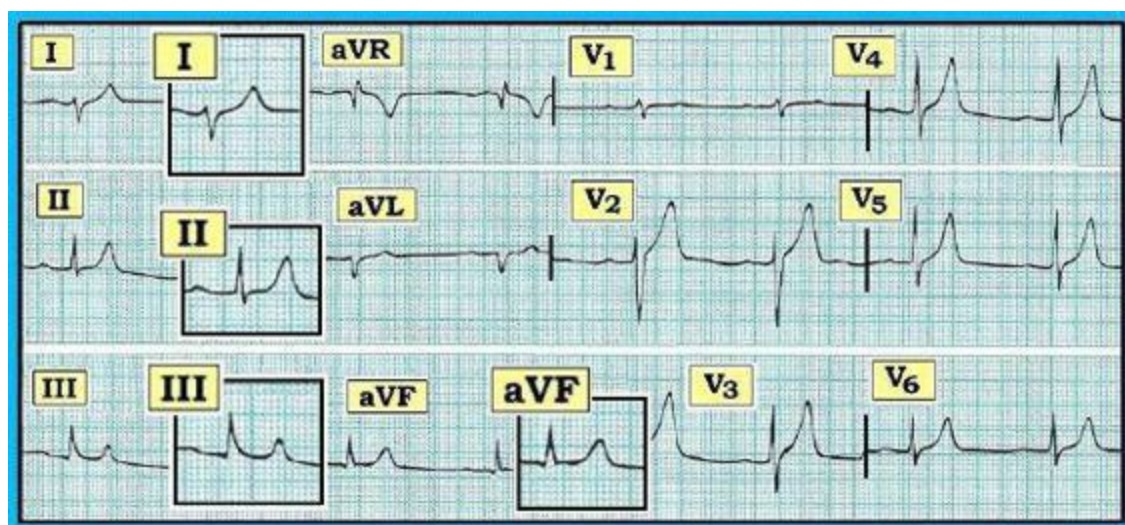


Figure 07.25-1: 12-lead ECG obtained from an otherwise healthy 30-year-old man. There is RAD.

Does the patient have LPHB? (*See text*).

Answer to Figure 07.25-1: Although there is *marked* RAD — We would not interpret this tracing as showing LPHB because: **i)** LPHB is rarely seen as an isolated conduction defect; and **ii)** The patient is an otherwise *healthy* 30-year-old man — which is a distinctly *unusual* setting for LPHB to occur.

- We would interpret this ECG as showing sinus arrhythmia; RAD; and peaked T waves with some J-point ST elevation that is most suggestive of an early repolarization pattern.
- Given the *considerable* amount of RAD seen here — the possibility of RVH (*Right Ventricular Hypertrophy*) should be contemplated. Assuming no murmur was heard on auscultation — the decision of whether or not to obtain an Echo could be made clinically.
- Perhaps if *new* RAD to this degree was seen in a patient with ongoing *acute* infarction — the cause might be LPHB (*as occurs in Figure 10.43-1*). However, given the setting described here — this is highly unlikely.
- On occasion — otherwise healthy adolescents and young adults may present with RAD *not* due to RVH or other underlying structural heart disease. We suspect this is the situation here.



The unfortunate clinical reality is that the ECG is just *not* very accurate as a diagnostic tool for determining chamber enlargement. Even in the best of hands — the *sensitivity* of ECG for detecting *LVH* does *not* exceed 60% (*although specificity may approach 90 to 95% when certain criteria are met*). Diagnostic accuracy for determining *RVH* (*Right Ventricular Hypertrophy*) and atrial enlargement is even less ... (See Sections 08.14-through-08.22; and 08.23-through-08.33).

- Echocardiography is far superior to the ECG for diagnosing enlargement of *any* cardiac chamber.
- Therefore — IF you truly *need* to know — *Get an Echo!*

08.1 – ECG Diagnosis of LVH: Simplified Criteria

There are *more than* 50 sets of criteria in the literature for ECG diagnosis of LVH. *None are optimal*. The *simplified criteria* for LVH we favor as providing what we feel is the *best* balance between sensitivity and specificity are shown in Figure 08.1-1.

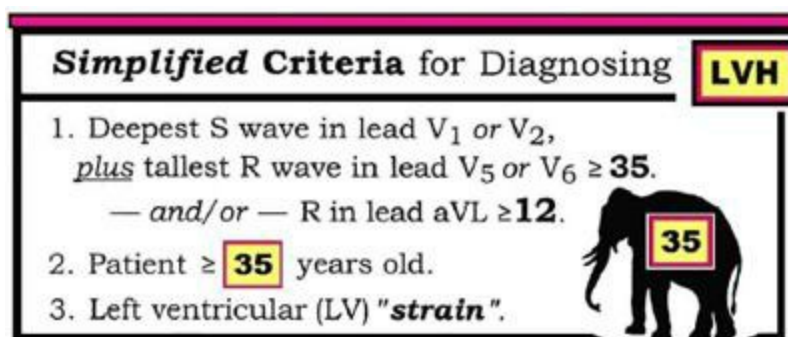


Figure 08.1-1: Simplified ECG criteria for LVH (See text).

KEY Points: The “beauty” of Figure 08.1-1 is that these *simplified* criteria *only* require recall of 2 numbers = “35” and “12”. In our experience — these 2 criteria *almost* always allow diagnosis whenever it is possible by ECG to determine that there is LVH.

- Only one voltage criterion (ie, 35 or 12) — needs to be satisfied for ECG diagnosis of LVH.
- Note that voltage criteria for LVH may *not* be **valid** for *younger patients*. This is because *younger* adults (*in their 20's*) often manifest increased QRS amplitude *without* true chamber enlargement. We suggest *not* using the voltage criteria in Figure 08.1-1 for patients *less* than 35 years old.
- The **accuracy** (*specificity*) for the ECG diagnosis of LVH can be **greatly increased** — IF in *addition* to satisfying one or more voltage criteria, ST-T wave changes of “*LV strain*” are *also* present (*Section 08.9*).
- The **Clinical History** helps in *assessing* for LVH. For example, it is important to appreciate

even before looking at the ECG itself — that the chance of *true* chamber enlargement is *greatly* increased IF the tracing is obtained from a middle-aged, African-American man with **longterm hypertension**. KEY POINT: A history of **underlying heart disease** (*heart failure, hypertension, cardiomyopathy, valvular disease, coronary artery disease*) — clearly **increases** the **likelihood** of LVH in any given patient.

08.2 – LVH: Physiologic Rationale for Voltage Criteria

The *physiologic* rationale for derivation of virtually *all* voltage criteria for LVH is simple. We illustrate this concept in **Figure 08.2-1**:

- Normal LV (Left Ventricle) activation is directed *away* from **right-sided leads** (ie, V1,V2) — and toward **left-sided leads** (aVL; V5,V6). As a result — leads V1,V2 *normally* manifest a predominant S wave.
- Since the LV is situated to the left and posteriorly in the thorax — normal LV activation generally results in a *predominant* R wave in *left-sided* leads aVL,V5,V6 (**Panel A** in Fig. 08.2-1). With LVH — there is *more* LV mass (*large red arrow* in **Panel B**). As a result — the S wave in V1,V2 becomes deeper; and the R wave in aVL *and/or* V5,V6 becomes taller.

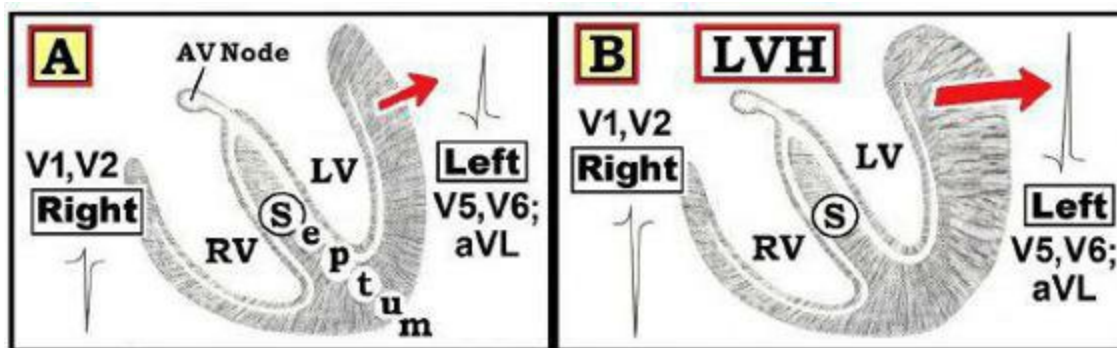


Figure 08.2-1: Rationale for derivation of LVH voltage criteria. There is *deepening* of the S wave in *right-sided* leads = V1,V2; and/or an *increase* in R wave amplitude in *left-sided* leads (ie, V5,V6, aVL).

08.3 – LVH: ECG Diagnosis using Lead aVL

In Section 03 — We discussed localization of **lead aVL** in the hexaxial lead system (*looking down at the heart from the left shoulder at an angle of approximately -30 degrees*). We then specified anatomic landmarks used for **precordial lead placement** when recording an ECG (Section 03.6). Awareness of this *relative* position of *unipolar* **lead aVL** (*looking down at the heart from the left shoulder*) — allows us to envision how lead aVL views the heart's electrical activity from a **higher perspective** than **precordial leads** (ie, V1,V2; V5,V6) that are placed on the chest.

- As a result — Use of the criterion for **R wave amplitude** in **lead aVL $\geq 12\text{mm}$** is likely to be *most helpful* when there is a **leftward axis** (*this aVL criterion may satisfy voltage for LVH despite sometimes minimal QRS size in V1,V2 and V5,V6*).

- Remember — Only 1 voltage criterion (35 or 12) is needed to satisfy “voltage for LVH”.

Figure 08.3-1: LVH using Lead aVL

We illustrate use of lead aVL for making the diagnosis of LVH by voltage in Figure 08.3-1. This ECG was obtained from an older adult with hypertension. R wave amplitude in lead aVL is clearly increased (*more than 3 large boxes — or greater than 15 mm*). However — there is no indication at all of voltage for LVH from assessment of QRS amplitude in the *precordial* leads (*deepest S in V1, V2 + tallest R in V5, V6 is very much less than 35 mm*).

- Since only 1 voltage criterion is needed — the presence of a *tall* R wave in lead aVL (≥ 12 mm) in Figure 08.3-1 satisfies voltage criteria for LVH.
- Note there is LAD — as is evident from the predominantly *negative* QRS in lead aVF with *positive* QRS in lead I. This supports the premise that lead aVL tends to be most helpful as a voltage criterion for LVH in the presence of a *leftward* axis.

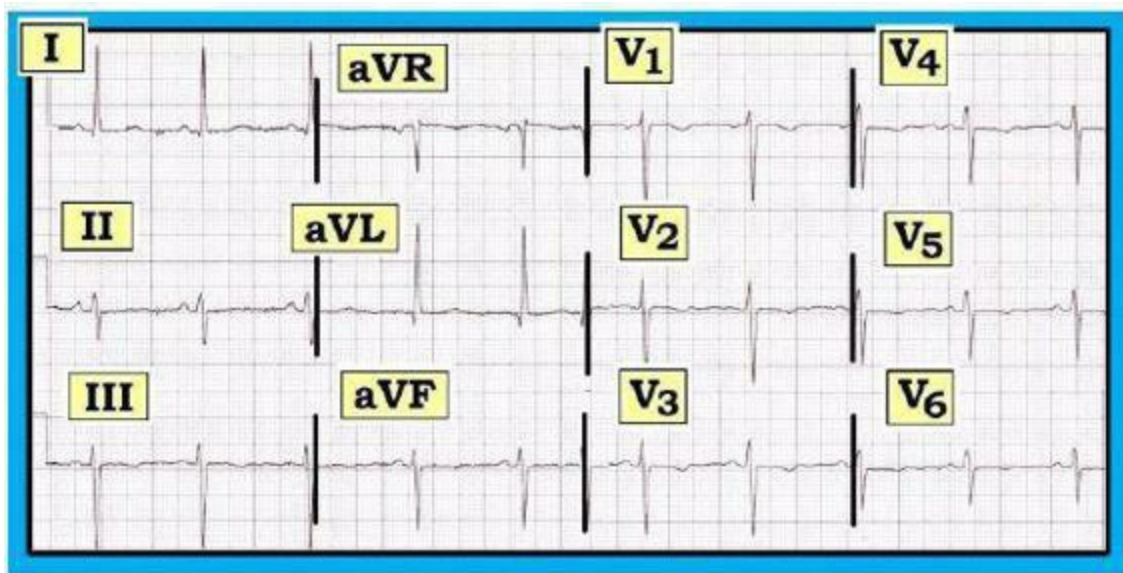


Figure 08.3-1: ECG obtained from an older adult with hypertension. Voltage criteria for LVH are satisfied by the *tall* R wave in lead aVL (≥ 12 mm) — despite relatively low voltage in the precordial leads (*See text*).

08.4 – FIGURE 08.4-1: Is there Voltage for LVH?

The ECG in Figure 08.4-1 was obtained from an otherwise healthy 29-year old man who was seen in the office for an “insurance physical”.

- *Is there LVH?*
- HINT: Feel free to review the *simplified* criteria for LVH in Figure 08.1-1 before answering.

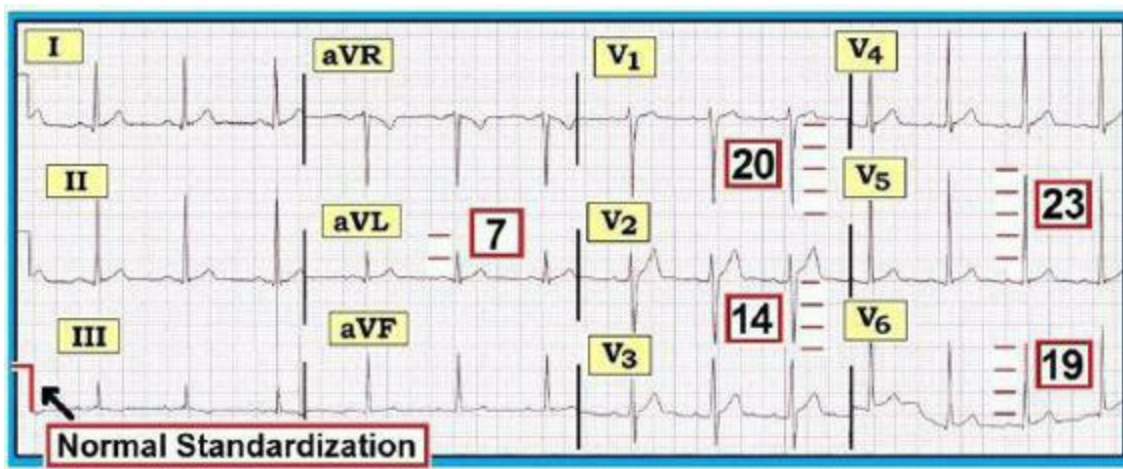


Figure 08.4-1: ECG from a 29-year old man. *Is there LVH?*

Answer to Figure 08.4-1: The ECG shows sinus arrhythmia and is essentially normal. *There is no LVH* — because this patient is *less* than 35 years old. Note that ST-T waves are unremarkable. There is *no* indication of LV “strain”.

- We illustrate how to assess for voltage by the *blow-up* of the 4 key precordial leads shown in **Figure 08.4-2**. As per the criteria detailed in **Figure 08.1-1** — one adds the *deepest* S wave in leads V1,V2 plus the *tallest* R wave in V5,V6. The sum = 43 (ie, 20 + 23=43) — but since the patient is *under* 35 years of age — voltage for LVH is not present (*Section 08.7*).

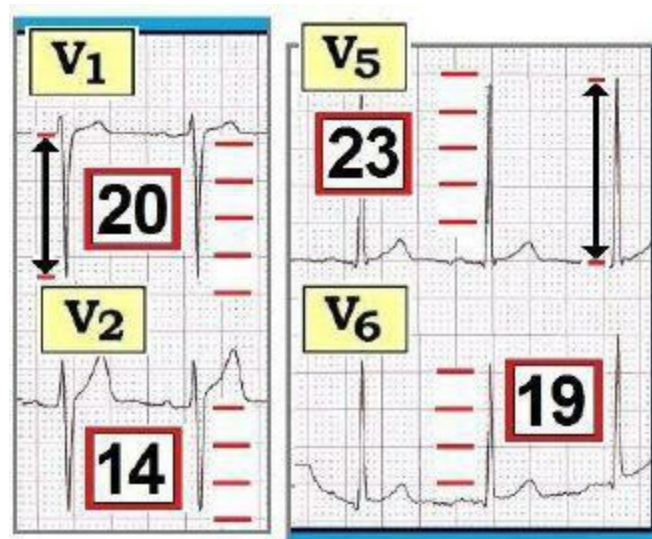


Figure 08.4-2: *Blowup* of selected precordial leads from **Figure 08.4-1** to illustrate how QRS amplitude is counted. The *deepest* S wave in V1,V2 is 20 (in lead V1) + the *tallest* R wave in V5, V6 (= 23 in lead V5) is >35 – BUT – because the patient is *less* than 35 years of age — voltage for LVH is not present.

08.5 – Standardization Mark: Is Standardization Normal?

Most of the time — the ECG will be set to **normal standardization**. Confirmation that this is the case is easily achieved by recognition of the **standardization mark** at the very beginning or end of the 12-lead recording (*black arrow at the onset of lead III in Figure 08.4-1*).

- **Normal standardization** is designated by a *rectangular* standardization mark that is **10 mm (=2 large boxes) tall**.
- On occasion — ECG complexes may be extremely large and extend *beyond* the space provided for one or more leads on the tracing. Selection of **half standardization** reduces waveform amplitude by half — with result that the *entire* complex in each lead will again fit on the ECG recording paper and be seen.
- These concepts are illustrated in **Figure 08.5-1** — in which the **Top Panel** is a *blow-up* of leads III and aVF from Figure 08.4-1.
- IF instead, this ECG was recorded at **half standardization** — *actual* QRS amplitude would be *twice* that shown = 10 vs 5 in lead III; and 26 vs 13 in lead aVF (**Lower Panel** in Fig. 08.5-1).

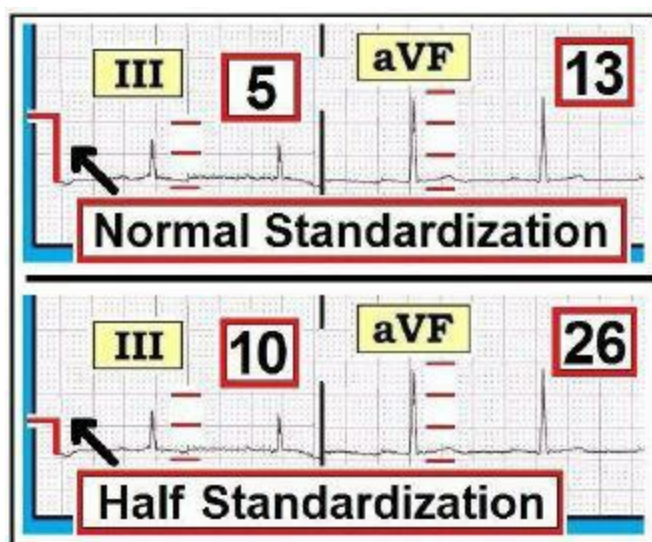


Figure 08.5-1: *Blowup* of Figure 08.4-1 to illustrate the appearance of a **normal** (*top*) and **half** (*bottom*) **standardization** mark. Actual voltage is *twice* that shown when the ECG is recorded at *half* standardization.

08.6 – LVH: Additional Voltage Criteria

No single voltage criterion will identify all patients with LVH. As a result — we occasionally turn to **additional voltage criteria**. We favor *any* of the following:

- A **deep S wave** (≥ 20 -25 mm) in lead **V1** or **V2**.
- A **tall R wave** (≥ 25 mm in **V5** — or ≥ 20 mm in **V6**).
- An **R wave** ≥ 20 in *any* inferior lead (**II**, **III**, or **aVF**).
- **Cornell Criteria** — LVH is present IF sum of **R** wave in lead **aVL** + **S** wave in lead **V3** is ≥ 20 mm (*female*) or ≥ 28 mm (*male*).

Bottom Line: In our experience, IF all you remember are the numbers '**35**' and '**12**' for **LVH voltage criteria** — then $\sim 90\%$ of the time when it is possible to diagnose LVH on the ECG of an **adult**, you will be able to do so!

- Satisfying any of the above **additional criteria** should help to pick up most of the remaining

~10%.

- Incorporating ***Clinical History*** and looking for “***strain***” or a strain “***equivalent***” pattern (Section 08.13) — will further refine and increase accuracy (*specificity*) of your diagnosis.
- Keep in mind that ***competing conditions*** (ie, *hyperkalemia, acute infarction, conduction defects, pulmonary disease*) — may *mask* ECG diagnosis of LVH.
- ***IF*** you *really* need to know about chamber size — ***Get an Echo***. The ECG is simply *not* optimally accurate.

08.7 – LVH: Voltage Criteria for Patients Less than 35

Young adults often have increased amplitude *not* due to true chamber enlargement. As a “***ballpark***” estimate for LVH voltage criteria when the patient is **35 years old** — We use sum of the *deepest* S wave in V1,V2 + *tallest* R wave in V5,V6 ≥ 53 . This number ‘53’ can be easily remembered — because it is the *reverse* of the number ‘35’.

- Look at the selected precordial lead sequence shown in **Figure 08.7-1** (previously shown in Fig. 08.4-2). Sum of the *deepest* S wave in V1,V2 (20) + *tallest* R wave in V5,V6 (23) = 43 mm.
- We were initially told (in Section 08.4) — that these leads were obtained from a 29-year old man. In this case — voltage criteria for LVH would *not* be met (*the patient is under 35 — and the voltage sum is less than 53*).
- On the other hand, ***IF*** this patient was over 35 — then voltage criteria for LVH *would* be satisfied (*since the voltage sum is more than 35*).

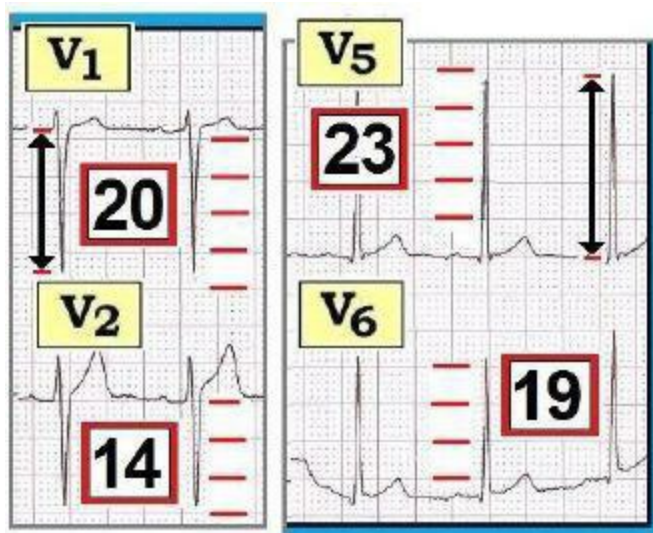


Figure 08.7-1: Selected precordial leads reproduced from Fig. 08.4-2. Is there voltage for LVH (See text).

KEY Point: There is no “magic” per se about becoming 35 years old and suddenly satisfying voltage criteria for LVH. The age “cutoff” is far from precise. That said, it is helpful to be aware that more often than not when a *healthy* adult under 35 manifests *increased* QRS amplitude but *without* ST-T wave changes of “strain” — most of the time there is *not* true chamber enlargement.

08.8 – FIGURE 08.8-1: Which Leads for What with LVH?

In an attempt to *expedite* ECG recognition of LVH — We suggest use of **Figure 08.8-1**. This figure highlights our two favored LVH voltage criteria (*Section 08.1*):

- LVH by “**35**” — We look for *deepest* S wave in V1,V2 + *tallest* R wave in V5,V6. If ≥ 35 = LVH.
- LVH by “**12**” — If the R wave in lead aVL ≥ 12 = LVH.
- We then look for LV “**strain**” or a strain “**equivalent**” (*Section 08.13*) — in one (*or more*) of the **left-sided leads** = I,aVL; V4,V5,V6.
- For **LAE** — Look at lead II (*notched P wave*) and/or lead V1 (*deep negative component to the P in VI*).
- For **RAE** (*not shown on this Figure*) — Use lead II (*Sections 08.19*).

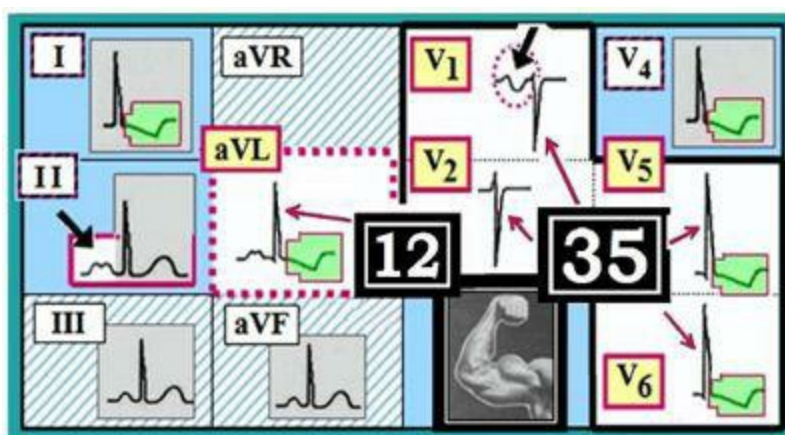


Figure 08.8-1: Chamber Enlargement: *Which leads for what?*

08.9 – LV “Strain”: ECG Recognition

It is difficult to define “*strain*”. We conceptualize the ECG picture of “*strain*” as — a pattern of **asymmetric ST-T depression** (*Figure 08.9-1*). Perhaps “strain” simply reflects that point of anatomic ventricular enlargement at which increased demand (*from chamber thickening*) outstrips blood supply — with *resultant* ST-T wave repolarization changes from *suboptimal* perfusion. Or *perhaps not*. That said — What counts clinically is ECG *recognition* of a “**strain**” pattern.

- Note how *downslope* of the ST segment in *Figure 08.9-1* is *slower* with “**strain**” (*arrow in Panel C*) — compared to the *more rapid return* of the ST segment to baseline.

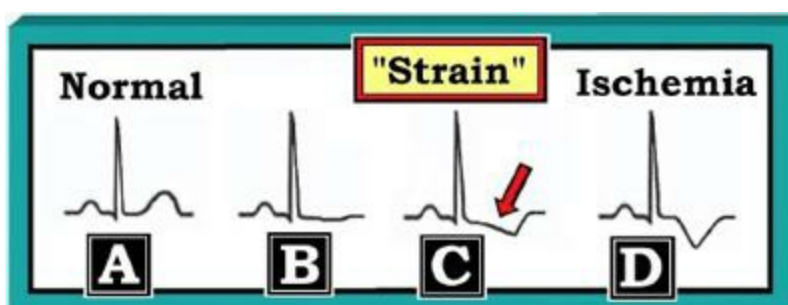


Figure 08.9-1: ST-T wave appearance of normal (**A**) — vs “strain” (**C**) *or a strain “equivalent”* (**B**) — vs ischemia (**D**).

Pattern Recognition: The concept of *pattern recognition* is essential to ECG diagnosis. Consider the following:

- With **ischemia** (*seen in Panel D of Figure 08.9-1*) — there will often be **symmetric T wave inversion** in two or more leads in a general lead area.
- **NOTE:** As one traces the path in *Figure 08.9-1* from a *normal* ST-T wave (*seen in Panel A*) — to the **asymmetric ST-T wave** typical of LV “strain” (*seen in Panel C*) — one passes through an **intermediate stage**, where the ST-T wave *flattens* with beginning ST depression (**Panel B**). We designate this intermediate stage as a **strain equivalent**.

Terminology: In general, when we use the term “**strain**” — We are referring to LV “strain” that develops in response to **Left Ventricular Hypertrophy (LVH)**.

- There is also **RV “strain”** — that typically develops in association with *marked RVH* or in response to acute *pulmonary embolus*. For now — *Think LV “strain”* when we use the *unspecified* designation of “strain” (*RV “strain” is discussed in Sections 08.28, 08.29*).

Which are the Leads that may show LV “Strain”?

We expect to see LV “strain” in leads that **look** at the **left ventricle**. These include one (*or more*) of the **lateral leads** I, aVL; and V4, V5, V6 (*highlighted by green boxes in Figure 08.8-1*).

- *Less often* — a typical LV “strain” pattern (*with asymmetric ST-T wave depression*) may also be seen in the **inferior leads** — especially if the patient has a relatively vertical (*inferior*) axis.
- LV “strain” — is generally not seen in *anterior* leads (*V1, V2, V3*). In contrast, **RV “strain”** (*as may occur with RVH or acute pulmonary embolus*) — typically is seen in either *anterior* or *inferior* leads (*See Section 08.29*).

FIGURE 08.9-2: Which Leads Show LV “Strain”?

The ECG in *Figure 08.9-2* was obtained from a patient with longstanding hypertension. There is **obvious voltage** for LVH (*very deep S wave in V2 + very tall R wave in V6*).

- **Which** of the 5 **lateral leads** manifest ST-T wave changes consistent with “strain”?
- Explain the ST-T wave depression seen in each of the **inferior leads** (*II, III, aVF*).

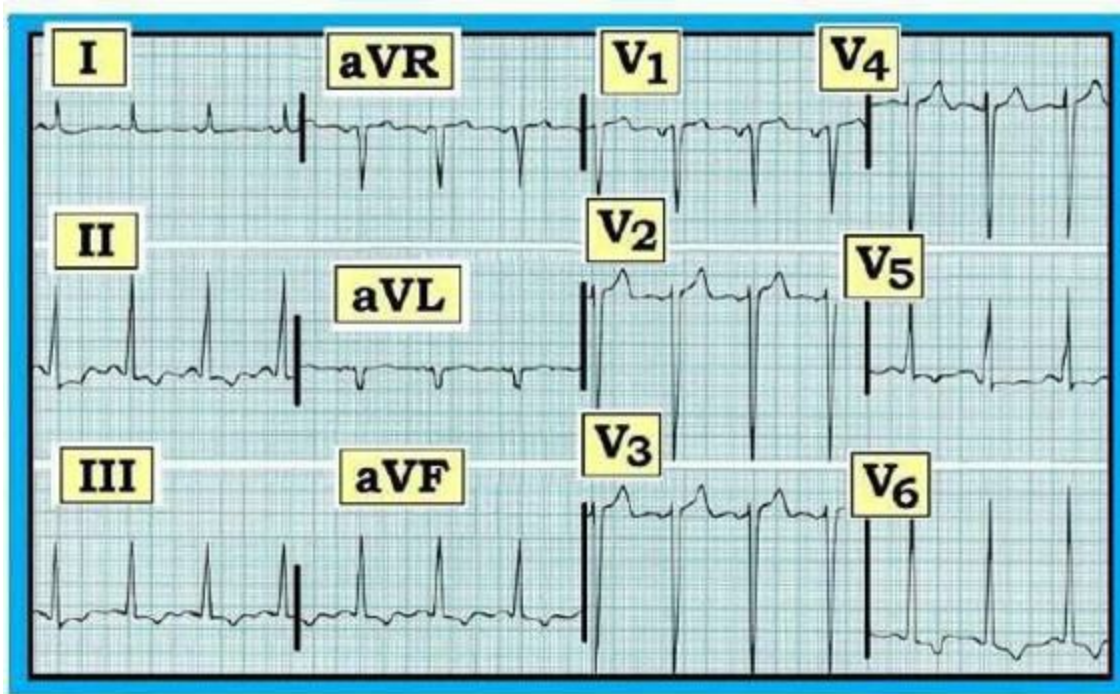


Figure 08.9-2: ECG from a patient with longstanding hypertension. Which leads show ST-T wave changes consistent with LV “strain”? (See text).

Answer to Figure 08.9-2: The rhythm is sinus. As stated — there is *obvious* voltage for LVH (very deep S wave in V2 + very tall R wave in V6).

- ST-T wave changes consistent with LV “strain” are clearly seen in **lead V6** of Figure 08.9-2. We would interpret the *flat* ST segment with slight T inversion in **lead V5** as a “**strain equivalent**” (Section 08.13). Neither aVL nor lead V4 manifest “strain” (upright T wave in these leads). The nonspecific ST-T wave flattening in lead I might be a strain “equivalent” — albeit *minimal* QRS amplitude in this lead makes this supposition less convincing.
- **NOTE:** Asymmetric ST-T wave depression in each of the **inferior leads** (II,III,aVF) in this tracing looks typical for LV “strain” (closely resembles **Panel C** in Fig. 08.9-1). Given the relatively vertical axis (about +75 degrees) — it is far more likely that these inferior ST-T wave changes reflect LV “strain” rather than ischemia (especially if the patient is not having new or recent chest discomfort). Thus, the ECG in Fig. 08.9-2 appears to represent an example of the occasional occurrence of LV “strain” in *inferior* as well as lateral leads.

Beyond-the-Core: NOTE in Figure 08.9-2 — that the **ST segment** is **elevated** in leads **V2,V3**.

- Is this likely to reflect an ACS (Acute Coronary Syndrome)?

Answer: As will be discussed in detail in Sections 09 and 10 — ST segment deviations (*elevation or depression*) are most often judged with respect to the *preceding* PR segment baseline. With this in mind — there unmistakably is *at least* 2 mm of ST elevation in leads V2,V3. That said — it is highly **unlikely** that this **anterior ST elevation** represents ACS because:

- There is *no mention* of chest discomfort in this patient with longstanding hypertension.

- In patients with LVH and “strain” — there will often be several mm of ST elevation in *anterior* leads as a **mirror-image** reflection of *lateral* precordial “strain”. Note that IF you *flipped over* the ECG complex in lead V6 of Figure 08.9-2 (*very tall R wave; ST-T changes of strain*) — that the result would be *identical* to what is seen in leads V2,V3 (*very deep S wave; mirror-image ST-T wave appearance of what was seen in V6*).
- **BOTTOM Line:** In addition to ST-T wave depression that may be seen in one or more of the *lateral* leads with LV “strain” — *slight* ST elevation (*with mirror-image appearance to the ST-T wave in lateral leads*) may also be seen in *anterior* leads.

08.10 – LV “Strain”: Voltage for LVH vs True Chamber Enlargement

The reason it is important to recognize “strain” — is that this finding *greatly* increases the *accuracy* of the ECG diagnosis of LVH.

- The presence of *increased voltage without* “strain” — has *low* specificity (50%) for *true* chamber enlargement (*especially if the patient is otherwise healthy and without underlying heart disease*). We interpret the finding of *increased voltage without* ST-T wave changes of “strain” as **“Voltage for LVH”**. This designation serves to acknowledge that despite an increase in QRS amplitude — *statistical* likelihood of true chamber enlargement is low.
- **NOTE:** On occasion there may be *typical* ST-T wave changes of LV “strain” — but without **voltage** for LVH. The ECG is simply not that sensitive for demonstrating increased QRS amplitude in patients with anatomic LV chamber enlargement. While we do not advise making a definitive ECG diagnosis of “LVH” in the absence of voltage criteria — We may nevertheless *suspect* LVH rather than ischemia in the presence of *asymmetric* ST depression IF: **i)** the history does *not* sound like there is cardiac chest pain; and **ii)** the patient has one or more conditions predisposing to LVH. Clearly — *Clinical correlation is needed*.

Assess the ECG in **Figure 08.10-1** for LVH. We previously encountered this tracing in Figure 08.4-1 — at which time we were told that it came from a 29-year old man. Despite sum of *deepest* S wave in V1,V2 + *tallest* R wave in V5,V6 being >35mm — the patient’s age (ie, *less than 35 years old*) meant that voltage criteria for LVH were not fulfilled.

- BUT — What IF instead of a 29-year old man, this ECG had been obtained from a **40-year old man**? Would there *now* be voltage for LVH?
- **NOTE:** ST-T waves are normal in all lateral leads in Figure 08.10-1 — such that there is *no* indication of LV “strain”.

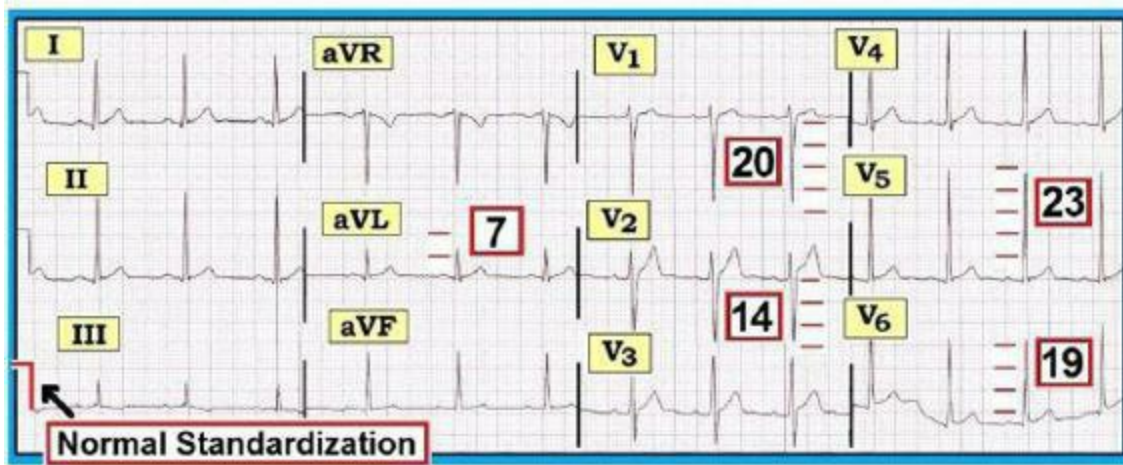


Figure 08.10-1: ECG previously seen in Fig. 08.4-1. Is there *voltage* for LVH? (See text).

Answer to Figure 08.10-1: IF instead of being 29, the patient was 40 — then the ECG in Fig. 08.10-1 would meet *voltage criteria* for LVH. That said — the *absence* of any ST-T wave indication of “strain” means that the chance of *true* chamber enlargement is still low. This is especially true if this 40-year old man was previously healthy with normal blood pressure.

08.11 – FIGURE 08.11-1: Is there True Chamber Enlargement?

Contrast the ECG just shown in Figure 08.10-1 — with the 6 precordial leads in **Figure 08.11-1**. These precordial leads were obtained from an *older* adult with *longstanding* hypertension.

- Is there LVH? What is the likelihood of *true* chamber enlargement?

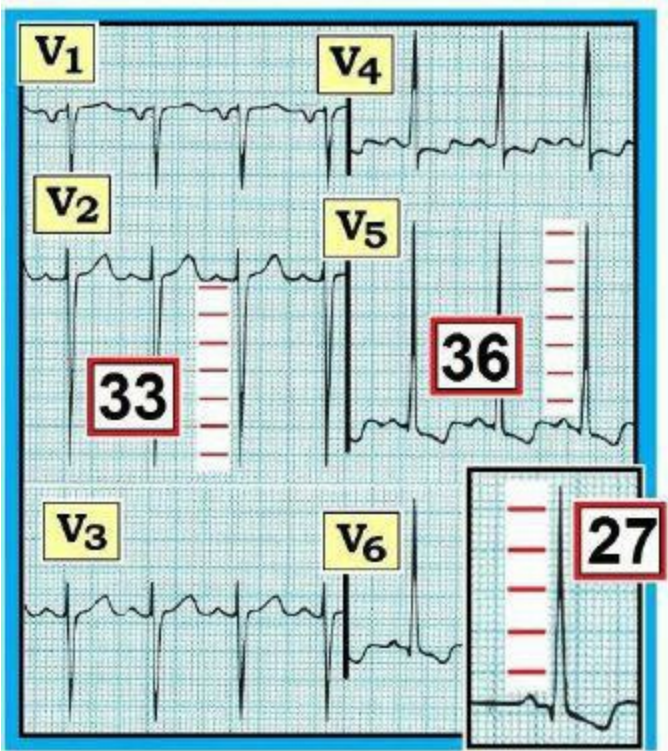


Figure 08.11-1: Precordial lead sequence obtained from an *older* adult with *longstanding* hypertension. What is the likelihood of *true* chamber enlargement?

Answer to Figure 08.11-1: QRS amplitude is *markedly* increased in the 6 precordial leads shown —

easily satisfying **voltage criteria** for LVH (Section 08.1). In addition — note ST-T wave changes consistent with LV “strain” in leads V4,V5,V6. *Specificity* for LVH has got to be *close to 100%*.

KEY Point: Once “strain” *and* voltage are *both* present — *specificity* for *true* chamber enlargement goes way up (to ~90% — and >95% if the patient has underlying heart disease). This is seen in Figure 08.11-1.

- ECG diagnosis of LVH *plus* “strain” is *not* a trivial one. The Framingham study demonstrated up to an **8-fold increased morbidity/mortality** in patients with longstanding hypertension when there was evidence of LVH *with* “strain” on their office ECG tracing.

08.12 – Can there be *both* LV “Strain” *and* Ischemia?

Clinically — *both* ischemia *and* “strain” may exist at the same time. Patients with LVH from longstanding hypertension may also have coronary disease. If anything — such patients are at *greater* risk of ischemia/infarction.

- The **ECG changes** of *either* ischemia *or* “strain” *may mask* the *other*. In some patients — *symmetric* T wave inversion may predominate. In others — J-point depression with *asymmetric* ST segment sagging may be more prominent. Distinguishing between *acute* ischemia *vs* “strain” may be extremely challenging (*if not impossible*) from review of a *single* ECG tracing.
- **NOTE:** When history *and* ST-T wave appearance suggest that *both* LV “strain” *and* ischemia are present — We *often* write the following on our interpretation: “**LVH *and* strain *and/or* ischemia. Suggest clinical correlation**”. This is precisely what we would write for interpretation of the precordial lead sequence shown in **Figure 08.12-1**.

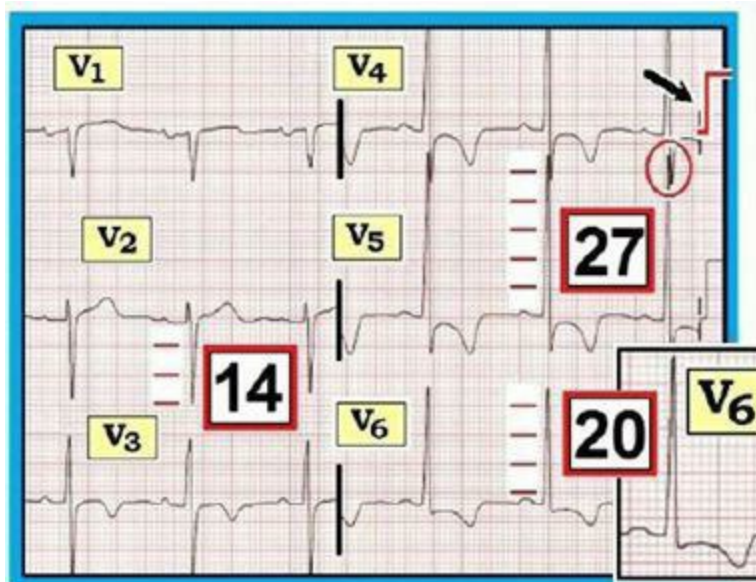


Figure 08.12-1: Precordial lead sequence obtained from an *older* adult with a history of coronary disease. We would *write* the following for our interpretation: **LVH *and* “strain” *and/or* ischemia** (See text).

Figure 08.12-1: Additional Points of Interest

- Note that **standardization** is **normal** in Fig. 08.12-1 (*arrow at the end of V4 indicating the rectangular standardization mark is 2 large boxes tall as it should be*).
- Note **slight variation** in **S wave depth** in lead **V2**. Such slight variation is common. We sometimes adjust for it by taking an *average* for amplitude in a given lead.
- Note **overlap** (*within the red circle*) of the **S wave** from **V4** with the **R wave** in **V5**. We could have eliminated this by recording the tracing at *half* standardization. That said — *most* of the time we prefer normal standardization. The point to emphasize is that caution is needed in determining *true* R wave amplitude in lead V5 of **Figure 08.12-1**.

KEY Points: Comparing Figs. 08.11-1 and 08.12-1:

Our interpretation of ST-T wave appearance in Figure 08.12-1 contrasts with that from Fig. 08.11-1. The best way to highlight this contrast is by *side-to-side* comparison of both tracings (**Panels A and B in Figure 08.12-2**):

- In both Panel A and Panel B — *deepest* S wave in V1,V2 + *tallest* R wave in V5,V6 easily satisfy **voltage criteria** for LVH.
- In Panel A — ST-T wave appearance in leads V5,V6 strongly suggests LV “**strain**” (*there is asymmetric ST depression with slow sagging downslope*).
- In contrast, for Panel B — ST segments are *coved* in V3-through-V6. In addition, there is 1-2 mm of J-point ST depression (*below the PR segment baseline*) in leads V4,V5,V6 — with **T wave inversion** that is *deep* and appears to be much *more symmetric* (*especially in leads V4,V5*). This appearance is *more* suggestive of **ischemia**.
- In particular — **lead V3 in Panel B** looks **ischemic** (*pure LV “strain” virtually never produces ST coving and symmetric T inversion this far anterior*).
- **Lead V6 in Panel B** — has an *intermediate* appearance between “strain” *and/or* ischemia. Given that this patient is “older” and has a history of heart disease and *decidedly* meets voltage criteria for LVH — We would interpret this tracing as, “**LVH and “strain” and/or ischemia.**” *Clinical correlation and comparison* with prior tracings would be needed to determine IF an *acute* ischemic process might be ongoing.

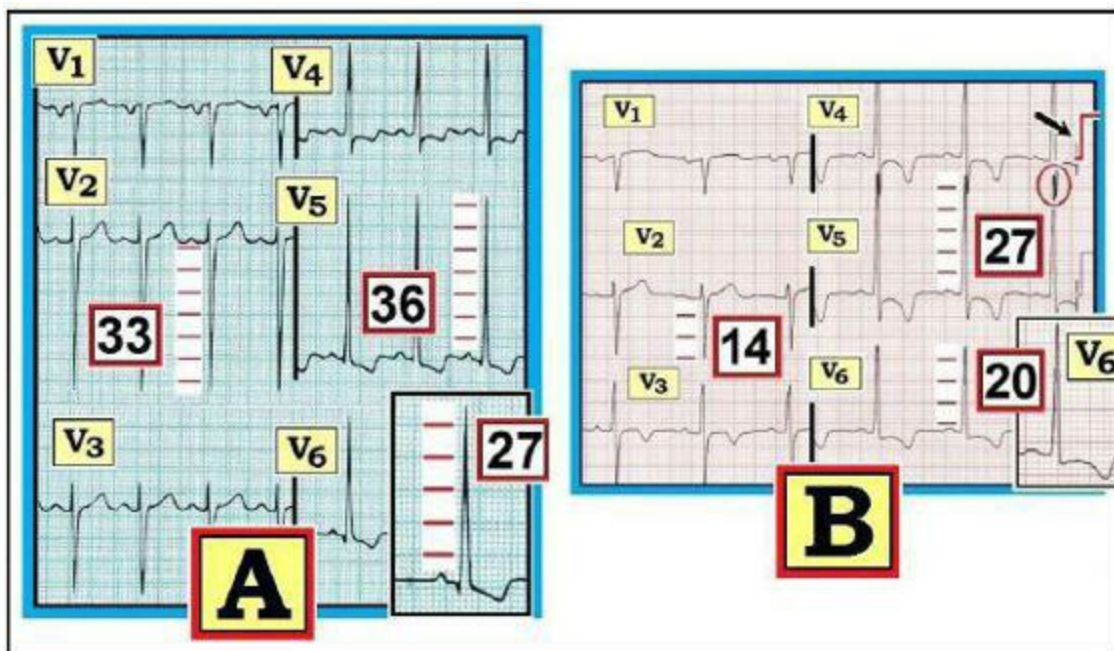


Figure 08.12-2: Comparison of the precordial lead sequence from Figure 08.11-1 (**Panel A**) with that from Figure 08.12-1 (**Panel B**). Voltage criteria for LVH are easily satisfied in each tracing. ST-T wave changes in **Panel B** are much more suggestive of LVH *with* ischemia *and/or* “strain” (See text).

Return to **Panel A** in Figure 08.12-2. Note that the J-point appears to be slightly *depressed* in leads V4-through-V6, and that there is subtle *ST coving* in the depressed ST segment in V4. Thus, although the overall picture in **Panel A** is clearly *more* suggestive of LVH with “strain” — We can not exclude the possibility of some component of ischemia.

- *Clinical correlation is needed.*
- Availability of *prior* tracings on this patient would help to determine whether J-point depression in **Panel A** is new or old. In the meantime — Interpreting ST-T wave changes in **Panel A** as consistent with “*strain and/or ischemia*” with need for *clinical* correlation is appropriate.

08.13 – Strain “Equivalent” Patterns: *Clinical Implications*

As discussed in Section 08.9 — an intermediate “*strain equivalent*” stage exists, in which the ST-T wave is *not* completely “normal” — but does not yet manifest full appearance of the *asymmetric* sagging ST segment characteristic of “strain”. We expand on this concept in **Figure 08.13-1**:

- Clinical implications of **LVH voltage** *plus* a “*strain equivalent*” (any of the 3 examples shown below in Figure 08.13-1) — are the same as they are for the fully developed pattern of “**strain**” (=greatly increased specificity for LVH — and a worse longterm prognosis).

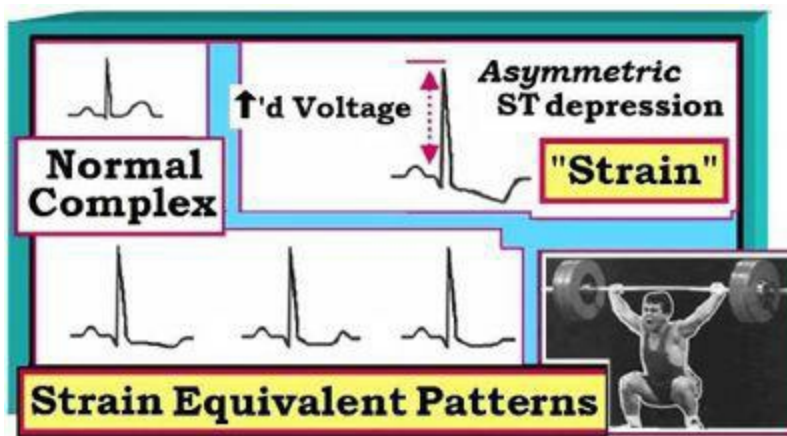


Figure 08.13-1: Examples of “strain equivalent” patterns (See the illustrative ECG shown below in Figure 08.13-2).

FIGURE 08.13-2: Is there LVH with “Strain”?

Apply the concept of recognizing an ST-T “strain equivalent” pattern to the precordial lead sequence shown in **Figure 08.13-2**, taken from a middle-aged man with hypertension.

- Are ECG criteria for LVH satisfied? If so — What is the likelihood of *true* chamber enlargement in Fig. 08.13-2?

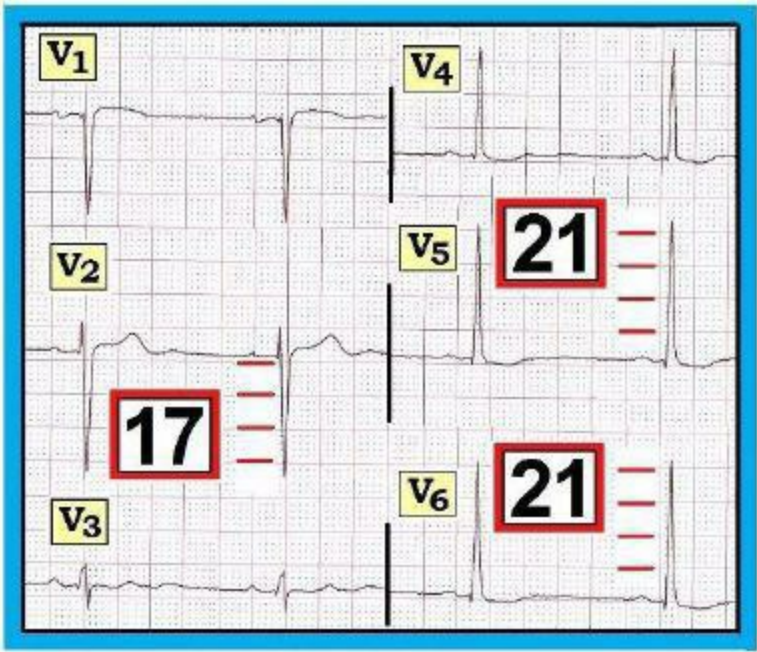


Figure 08.13-2: Precordial lead sequence from a patient with hypertension. Is there LVH? What is the likelihood of true chamber enlargement? (See text).

Answer to Figure 08.13-2: Voltage criteria for LVH are satisfied — as sum of *deepest* S wave in V1,V2 + *tallest* R wave in V5,V6 is >35 (ie, 17 + 21 = 38).

- The ST-T wave segment in *lateral* leads V4,V5,V6 is not normal. Instead — there clearly is flattening of the ST segment and T wave, with a hint of ST depression. That said — a fully developed pattern of LV “strain” is not seen.
- **Impression:** The ST-T wave in leads V4,V5,V6 of Figure 08.13-2 closely resembles the 3rd

“strain equivalent” pattern shown in the lower panel of **Figure 08.13-1**. In view of the history (*hypertension*) and more than adequate voltage for LVH — we would write **“LVH”** (*not just ‘voltage’ for LVH*) on our interpretation. The likelihood of *true* chamber enlargement is high.

08.14 – Atrial Enlargement



The ECG is *neither* sensitive *nor* specific for **atrial enlargement** (*Echo is far more accurate*). That said — there *are* times when the ECG may provide *suggestive* clues which clinically are very helpful ([Figure 08.16-1](#)):

- Recognition of **LAA** (*Left Atrial Abnormality*) — may **support** the ECG diagnosis of **LVH** (*especially in the presence of underlying BBB*).
- Recognition of **RAA** (*Right Atrial Abnormality*) in association with other ECG signs — may strongly **suggest** **RVH** (*and/or pulmonary hypertension*). It may also suggest acute pulmonary embolus.
- ECG signs of **multichamber enlargement** (*at least LVH, LAA, RAA — and possibly also RVH*) — when seen in a patient with underlying heart disease **suggests cardiomyopathy**.

08.15 – Terminology: *Enlargement* vs *Abnormality*?

Many clinicians use LAA/RAA — and LAE/RAE *interchangeably*. We prefer the terms, “**LAA**” and “**RAA**” (*where the “A” stands for Abnormality*) in acknowledgement that an **abnormal-looking P wave** on ECG does *not* always mean true *atrial* chamber enlargement. It simply means a *different-than-usual* P wave appearance.

- **Other Reasons** for **Abnormal P Waves** — include body habitus (*tall, peaked P waves may be seen in slender individuals with a vertical axis*); increased atrial pressure (*ie, from heart failure*) and intra-atrial conduction defects.
- Clinical context will usually suggest whether RAA/LAA is likely to reflect *true* atrial “enlargement” — or something else.
- If we *know* true atrial chamber **Enlargement** is likely (*because of marked LVH or RVH on ECG in a patient with underlying heart disease*) — then we may switch to the LA“**E**” or RA“**E**” designation. Otherwise — we generally prefer to use the terms LA“**A**” or RA“**A**”.
- **NOTE:** The atria do not “*hypertrophy*”, in that their walls don’t thicken. Instead — the atria enlarge (*dilate*).

08.16 – **FIGURE 08.16-1: ECG Criteria for RAA/LAA**

ECG Criteria for the diagnosis of RAA and LAA are summarized pictorially in **Figure 08.16-1**. Note that the 2 leads used to assess for atrial abnormality are lead II and lead V1.

- We explore the *physiologic* rationale for these ECG criteria in Section 08.17.
- We discuss specific ECG findings suggesting RAA and LAA in detail in Sections 08.18-through-08.20.

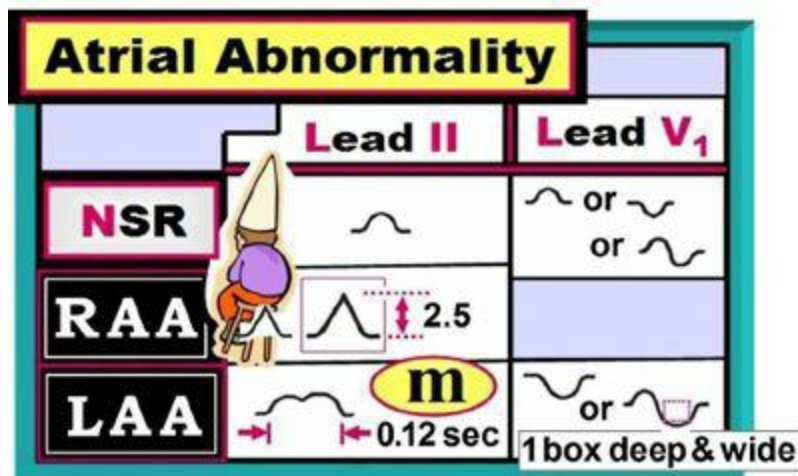


Figure 08.16-1: ECG signs of atrial abnormality/enlargement (See text).

08.17 – Physiologic Rationale for Normal P Wave Appearance

Physiologic rationale for the ECG signs of atrial enlargement is based on: **i)** *anatomic* location of **lead V₁** (the V₁ electrode lies over the RA = right atrium) — and — **ii)** *relative* direction of RA and LA depolarization with respect to the SA node (**Figure 08.17-1**).

- With **NSR** (*Normal Sinus Rhythm*) — the impulse begins in the **SA Node** — which lies in the upper part of the right atrium (**Panel A** in Fig. 08.17-1). **Activation** of the **RA** will therefore be seen as *approaching* lead V₁ — whereas activation of the **LA** will be seen as *moving away* from lead V₁ (red arrows in **Panel B**). This explains why in theory — the **P wave** in lead V₁ should normally be **biphasic**, with an initial *positive* component (as the RA is depolarized) — followed by a *negative* component (as the LA is depolarized).
- In Practice — the **normal P wave** in lead V₁ may be positive, negative or biphasic (as is shown under NSR in Fig. 08.16-1).
- The reason the **P wave** is normally **upright** in lead II with sinus rhythm is shown in **Panel C** of Figure 08.17-1. Summation of RA and LA activation is seen as *moving toward* lead II at +60 degrees (red arrows in **Panel C**).

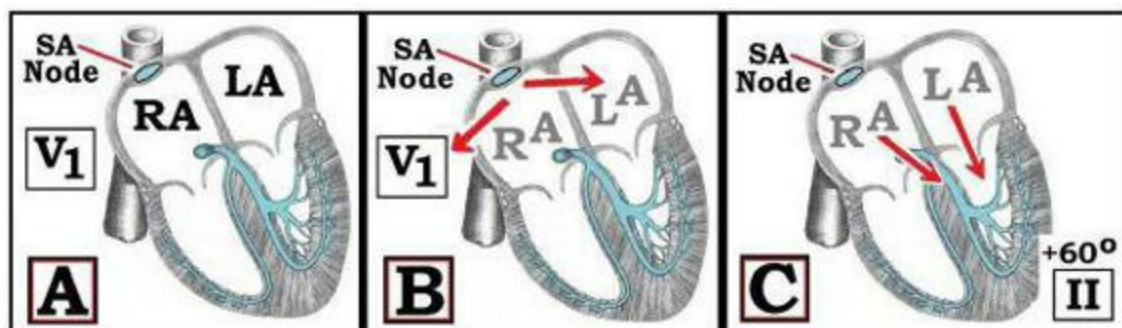


Figure 08.17-1: Physiologic basis for why with **NSR** (*Normal Sinus Rhythm*) — the P wave in lead V₁ may be positive, negative or biphasic (**Panel B**) — whereas in lead II, the *normal* P wave will be upright (**Panel C**).

08.18 – A Closer Look: The P Wave with Normal Sinus Rhythm

Appreciation of the concepts illustrated in [Figure 08.17-1](#) facilitates an understanding of normal sinus P wave appearance in leads II and V1 — as well as an understanding of what happens to P wave appearance in these same leads with RAA/LAA.

- With **NSR** (***N**ormal **S**inus **R**hythm*) — the P wave in lead II should be upright ([Figure 08.18-1](#)). However — the P wave may *normally* be upright, negative or biphasic in lead V1.
- Realize that IF there are 2 components to the P wave in lead V1 — the 1st component (*which will be positive*) represents RA depolarization (*since the sinus node is in the RA*). The 2nd component (*which is negative*) — represents LA activation. It is this 2nd (*negative*) component that gets larger when there is LAA ([Section 08.20](#)).
- **KEY Point:** The finding of a *negative* P wave in lead V1 is not abnormal. It does not necessarily mean the left atrium is large IF this negative component to the P wave is relatively small ([Section 08.20](#)).
- **Clinical Note:** Be aware that *minor* deviation from correct lead placement may alter P wave appearance in lead V1. *Despite* best intentions — the clinical reality is that precordial lead misplacement is relatively common.

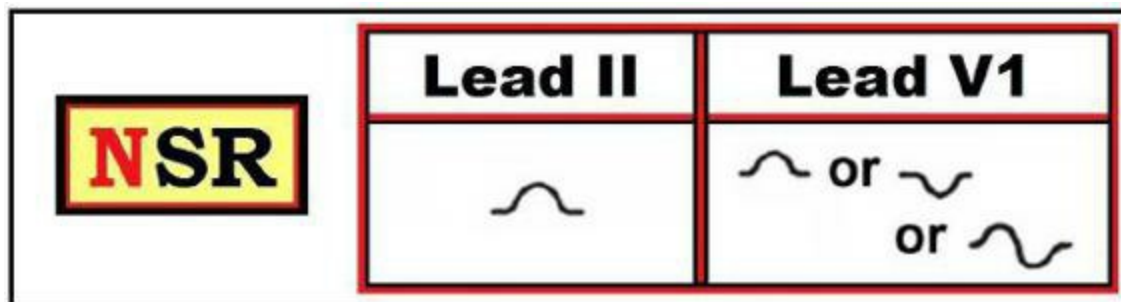


Figure 08.18-1: ECG appearance of the *normal* P wave in leads II and V1. Note that with sinus rhythm — the *normal* P wave in lead V1 may be upright, negative or biphasic.

08.19 – ECG Diagnosis of RAA: P Pulmonale

RAA (***R**ight **A**trial **A**bnormality*) — is diagnosed by the finding of tall **Peaked** and **Pointed** P waves in the **Pulmonary** leads ([Figure 08.19-1](#)). Because patients with COPD often have low diaphragms (*and therefore an inferior axis*) — we think of the **inferior leads** (II,III,aVF) as the “pulmonary” leads.

- The P wave should be *half* a large box tall (=2.5mm) — in *at least* one of the *inferior* leads for RAA.
- IF the **P wave** in one or more **inferior leads** looks “*uncomfortable to sit on*” — Think **RAA!** = **P Pulmonale**.
- Most of the time — Lead V1 will not be helpful in ECG diagnosis of RAA.
- *Beyond-the-Core:* On occasion — RAA may be suggested by P wave appearance in lead V1 IF the *positive* component of the P wave is pointed. This is true even if this pointed positive component is not quite 2.5 mm tall.



Figure 08.19-1: ECG criteria for **RAA** (*P pulmonale*). Most commonly — ECG diagnosis of RA is made from P wave appearance in lead II (*tall, peaked and pointed P wave at least 2.5 mm tall*). The P wave looks, “*uncomfortable to sit on*”. On occasion — RAA may be suggested by the finding of a *pointed* positive component to the P wave in lead V1.

08.20 – ECG Diagnosis of LAA: *P Mitrale*

LAA (*Left Atrial Abnormality*) — is diagnosed by finding an **m-shaped** (*notched*) and *widened* P wave (≥ 0.12 second) in a "**mitral**" lead (*I, II, aVL*) — *and/or* a deep *negative* component to the P wave in lead V1 = **P Mitrale** (**Figure 08.20-1**).

- The reason the P wave becomes *wider* with LAA is that the left atrium is activated *after* the right atrium (*since the SA Node is in the RA*). It is *addition* of this LA component to the P wave that produces the notch/extra 'hump' to form the "M" in lead II.
- The best ECG sign of LAA is the finding of a **deep, negative component** to the **P wave** in lead **V1** — that is *at least 1 little box deep or* wide.
- We generally **undercall** LAA — because of the overall *poor* specificity the ECG has for atrial enlargement. Remember that *some* negativity to the P wave in lead V1 is a *normal* finding! Therefore, it is best *not* to even contemplate an ECG diagnosis of "LAA" — *unless* the *negative* component to the P wave in lead V1 is definitely *at least 1 little box deep and/or* wide.

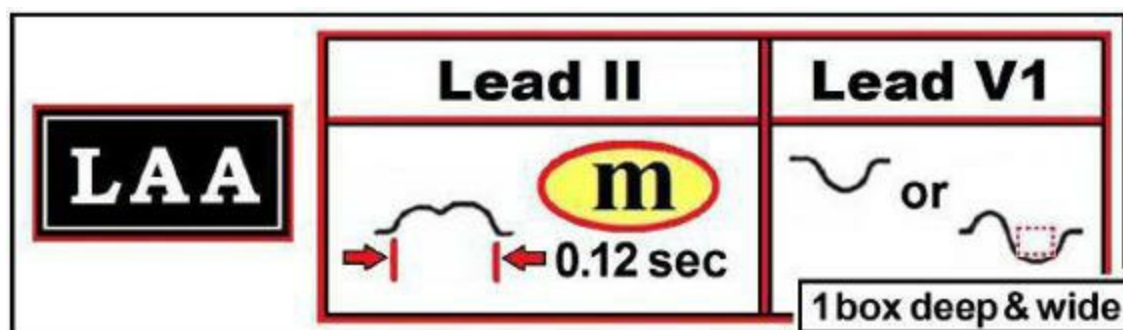


Figure 08.20-1: ECG criteria for LAA (*P mitrale*) in lead II and lead V1. Look for a notched (*longer*) P wave in lead II — *and* a deep *negative* component to the P wave in lead V1 (*that is at least 1 little box deep and/or wide*). It is best to *undercall* the ECG diagnosis of LAA (*See text*).

08.21 – FIGURE 08.21-1: *Is there ECG Evidence of RAA/LAA?*

Assess the ECG in **Figure 08.21-1** for RAA and LAA. Keep in mind that this tracing was obtained from an otherwise healthy and asymptomatic 27-year old man who was seen in the office for an insurance physical.

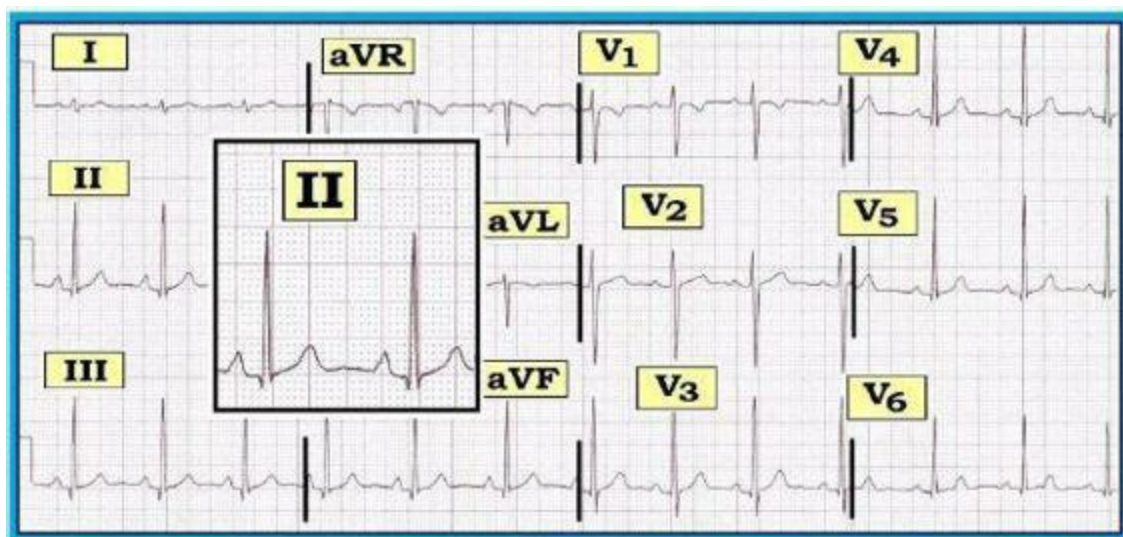


Figure 08.21-1: ECG obtained from a healthy 27-year old man. Is there ECG evidence for RAA/LAA? (*See text*).

Answer to Figure 08.21-1: There is sinus arrhythmia. All intervals ($PR/QRS/QT$) are normal. The axis is *vertical* but still within the normal range of zero to +90 degrees. Given the *near* isoelectric QRS complex in lead I (*which manifests no more than a minimal net positive deflection*) — We estimate the mean QRS axis to be about +80 degrees.

- There is *no* LVH — as the patient is *less* than 35 years of age (*Section 08.7*).
- There is **RAA** (*tall peaked and pointed P waves in the ‘pulmonary leads’ — with the P wave in lead II ≥ 2.5 mm*).
- There is *no* LAA — as the P wave is *not* notched, and there is *no* negative component to the P wave in lead V1.
- Regarding **QRST Changes**: There are small *septal* q waves in multiple leads; transition occurs *between* lead V2-to-V3 (*which is normal*); and ST-T wave changes are unremarkable.

Impression: This ECG is probably *not* abnormal — given that it was obtained from an otherwise healthy and asymptomatic 27-year old man. Use of the term, “**RAA**” — allows us to indicate that the P wave is larger than is normally seen without “*labeling*” this patient as having right atrial pathology. Instead — it is much more likely that the vertical axis and peaked inferior P waves seen here reflect a ***normal variant*** in an otherwise healthy young adult with a *vertical* ECG axis and an anatomically *normal* right atrium.

08.22 – FIGURE 08.22-1: *Is there ECG Evidence of RAA/LAA?*

The ECG in **Figure 08.22-1** was obtained from an older adult with heart failure. Assess this tracing for ECG evidence of chamber enlargement.

- What clinical diagnosis is suggested by the ECG picture shown here?

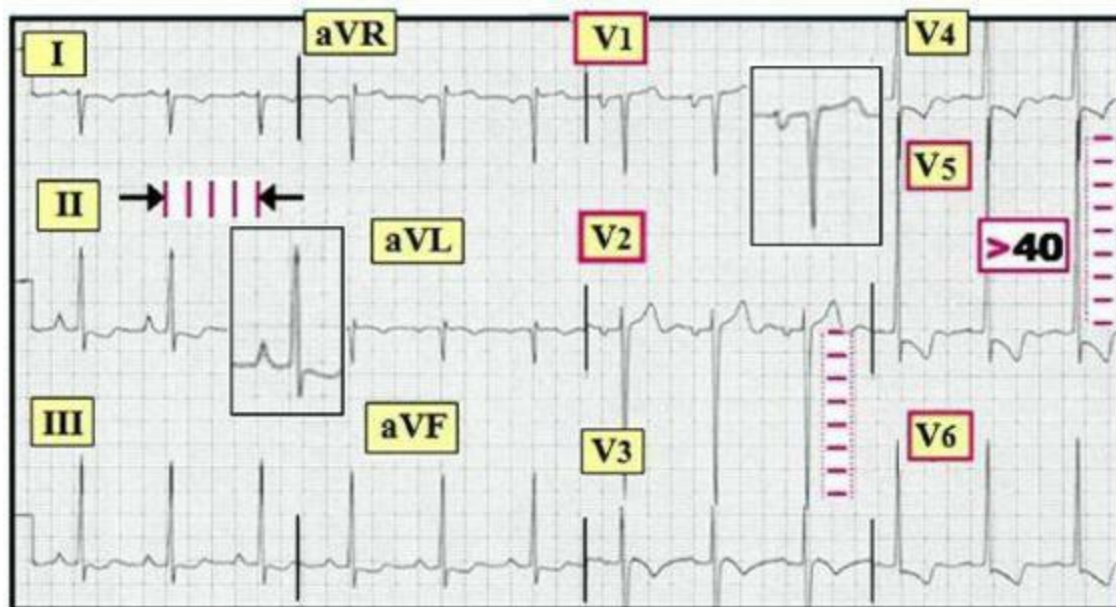


Figure 08.22-1: ECG obtained from an older patient with heart failure. Which cardiac chambers are enlarged? (*See text*).

Answer to Figure 08.22-1: The rhythm is sinus. The PR, QRS and QT intervals are normal. There is **RAD** (*Right Axis Deviation*) — as diagnosed by the markedly negative net deflection of the QRS in lead I (*Section 07.4*).

- There is **RAA** (*tall peaked and pointed P waves in the ‘pulmonary leads’ — with the P wave in lead II at least half a large box tall*).
- There is **LAA** (*deep negative component to the P wave in lead V1*).
- There is **LVH** (*very deep S waves in V1, V2 + very tall R waves in V5, V6*). ST-T wave changes are consistent with LV “**strain**” and/or **ischemia** (*Section 08.12*).
- There is *also* **probable RVH** (*marked RAD + RAA*).
- **PEARL:** The presence of **multichamber enlargement** (*at least 3 chambers*) in a patient with heart failure is *strongly* suggestive of **cardiomyopathy**.



RVH/*Pulmonary Disease*

Detection of right ventricular enlargement in adults by ECG criteria is often *exceedingly* difficult. This is because the LV (*Left Ventricle*) is normally so much larger and *thicker* than the RV (*Right Ventricle*) in adults — that it masks even moderate increases in right ventricular chamber size. As a result, many patients with **RVH** (*Right Ventricular Hypertrophy*) will *not* be identified — IF assessment for chamber enlargement is limited to obtaining an ECG (*an Echo is needed to know for sure*).

- **NOTE:** In contrast to adults — ECG diagnosis of **RVH** is often surprisingly *easy* in **children** with congenital heart disease (*because relative size of the RV compared to the LV is not nearly as different as it is in adults*).

08.24 – ECG Diagnosis of RVH: *Simplified Criteria*

Development of RVH may lead to a number of ECG findings. We list these in **Figure 08.24-1**. *None* of these findings alone will accurately predict *anatomic* RV enlargement. Instead — *Several* criteria are needed.

- Think of the ECG diagnosis of **RVH** as similar to making a "*detective*" diagnosis. *No single clue solves the mystery*. With regard to ECG interpretation — *No single finding* in **Figure 08.24-1** is diagnostic. Instead — determination of RVH is made by **deduction** (ie, *from identifying a combination of the ECG findings listed in Fig. 08.24-1*):


Findings Suggestive of RVH in Adults:	
	1. RAD or <i>indeterminate</i> axis
	2. RAA (which often <i>accompanies</i> RVH!)
	3. Incomplete RBBB (<i>or</i> an rSr' in lead V ₁)
	4. Low voltage (esp. with emphysema)
	5. Persistent precordial S waves
	6. "Strain" in right ventricular leads (which are V ₁ , V ₂ , V ₃ — <i>or</i> — II, III, aVF)
	7. Tall R wave in lead V ₁

Figure 08.24-1: ECG criteria for RVH. A *combination* of at least *several* of these criteria is needed for accuracy (See text).

Clinical NOTE: Pulmonary disease *without* frank RVH is common in longterm smokers. Progression to **cor pulmonale** (*in which there is frank RVH*) — represents a relatively *late* stage in the process. Careful search for the ECG findings in **Figure 08.24-1** may provide clues to *either* longterm pulmonary disease and/or associated RVH:

- Consider **Pulmonary Disease** — IF you see 2 or more of the first 5 criteria listed in Figure 08.24-1 (*especially IF the patient is a known smoker or has other known lung problems*).
- Suspect pulmonary disease **plus** RVH — IF in addition you also see a ***tall R wave in lead V1*** (*with or without ST-T wave changes of RV “strain”*).
- By Definition — By the time you see clear **ECG evidence** of RVH in an adult — the extent of **RVH** is usually ***marked*** (*the patient almost always has end-stage COPD and/or pulmonary hypertension*).

08.25 – ECG Diagnosis: *Review of Specific RVH Criteria*

While *no single finding* in Figure 08.24-1 *by itself* is enough to diagnose RVH — seeing ***several criteria*** on a *single tracing* is ***very suggestive*** of **RVH** — especially when seen in a *likely* clinical setting (ie, *COPD, longterm asthma, right-sided heart failure, pulmonary hypertension*). Over the next few sections — We review specific ECG criteria for RVH:

- **RAD** (*Right Axis Deviation*) — is highly suggestive of RVH when seen in a *likely* clinical setting and in association with *other* criteria listed in Figure 08.24-1. Few other conditions produce RAD. This is why we strongly ***suspect*** **RVH** is also present in the patient with cardiomyopathy whose ECG was shown in Figure 08.22-1. In the *clinical* context of heart failure with evidence of multi-chamber enlargement on ECG (*RAA/LAA/LVH*) — the markedly *negative* QRS complex in lead I of Fig. 08.22-1 (*which we reproduce below in **Figure 08.25-1***) suggests there is *also* enlargement of the 4th cardiac chamber (= *RVH*).
- **RAA** — It is usually easy to recognize the tall, **peaked**, **pointed** **P** waves of RAA (*Section 08.19*). **PEARL**: Only one condition produces RAA without at the same time producing RVH (= *tricuspid stenosis*). Therefore — identifying **RAA** on ECG is an ***indirect sign*** that **RVH** is ***very likely***. This was the case for the patient with cardiomyopathy whose ECG was shown in Fig. 08.22-1 (*reproduced below in **Figure 08.25-1***).

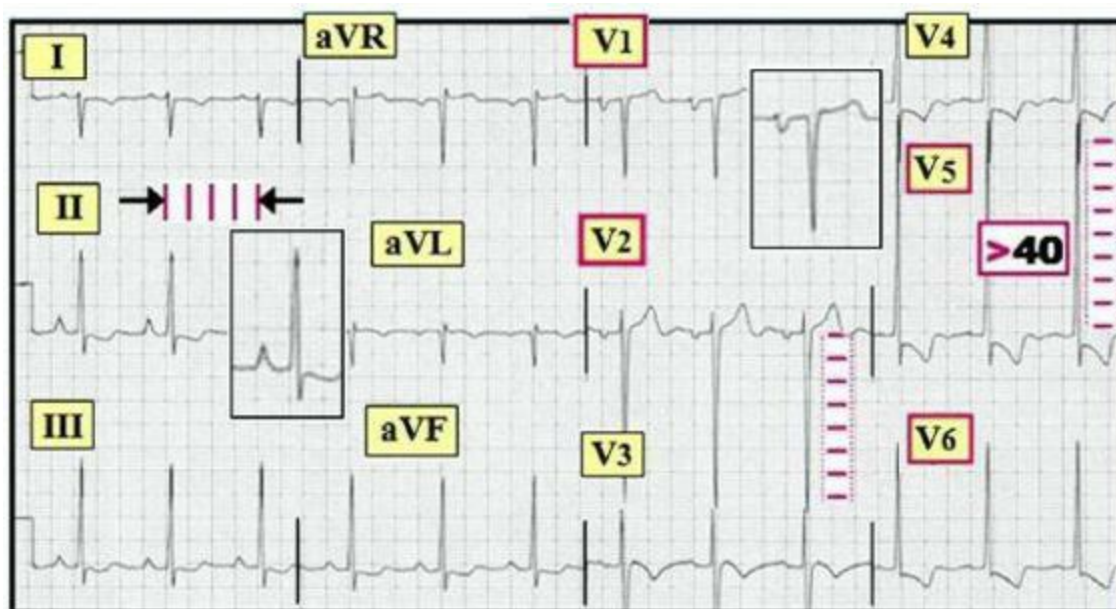


Figure 08.25-1: This ECG is *reproduced* from Fig. 08.22-1. The patient has a history of heart failure. The ECG shows **RAA** (*tall, peaked P wave in lead II*); **LAA** (*deep negative component to the P in VI*); and **LVH** with “strain” *and/or* ischemia (*markedly increased QRS amplitude in precordial leads with ST-T wave changes in V3-through-V6*). We strongly suspect a **cardiomyopathy with** enlargement of the 4th cardiac chamber (= **RVH**) because *in addition to* LAA/LVH: **i) RAA** is *also* present; and **ii)** there is otherwise unexplained **marked RAD** (*See text*).

08.26 – RVH: Review of Additional Criteria

Some findings in Figure 08.24-1 are more suggestive of RVH *and/or* pulmonary disease than others. The findings we describe below are *less* specific when seen in isolation — but they may be *diagnostic* when they occur in combination (*as we demonstrate in the next few sections*). Remember — *Not all findings are seen in every patient*.

- **Indeterminate Axis** — Alterations in lung volume with emphysema often lead to *rightward and posterior* axis deviation. As a result — *net* QRS deflection in *both* leads I and aVF may become negative. **PEARL:** IF ever you see an *indeterminate* axis — *Think RVH/COPD/Obesity* (*Section 07.8*).
- **IRBBB** (*rSr' in VI*) — The presence of an **r'** (*r prime*) in **lead V1** suggests that *terminal* electrical activity is directed toward the right. While this ECG sign is often benign and commonly seen as an *isolated* finding in otherwise *healthy* individuals (*Section 05.18*) — it supports the diagnosis of pulmonary disease/*possible* RVH if seen in association with *other* suggestive findings.
- **Persistent S Waves** — R wave amplitude normally increases as one moves across the precordial leads (*as electrical activity moves toward the left where the larger LV lies*). R wave amplitude usually peaks (*is tallest*) in V4 or V5 — and then drops off (*in V5,V6*). Normally, there is *not* any S wave at all in V5,V6 — since by this time in the depolarization process all electrical activity is traveling leftward. IF *more* than tiny **S waves** are **still present** in **V5,V6** — this implies significant *rightward* activity is still ongoing (*Think RVH/COPD/large body habitus*). That said — this finding is *not* present in Figure 08.25-1 (*although a ~5mm S wave is seen in lead V5 — no S wave at all is seen in lead V6*).
- **Low Voltage** — Air is *not* a good conductor of electricity. The large emphysematous chest of a patient with COPD dampens (*reduces*) voltage. Technically — “**low voltage**” is defined as QRS amplitude ≤ 5 mm (ie, ≤ 1 large box) in all 6 limb leads (I,II,III; aVR,aVL,aVF). That said — we also use “**low voltage**” as a *relative term* when **overall QRS amplitude** *subjectively* appears to be reduced. When you see “**low voltage**” — *Think COPD* (*although low voltage may also be seen in hypothyroidism; obesity; pneumothorax; pericardial effusion; or as a normal variant*).

NOTE: Using the above definition for **low voltage** (ie, *the QRS being ≤ 5 mm in all 6 limb leads*) — this ECG finding is *not* all that common. When *low voltage* is seen — it is a **nonspecific finding**. That said — IF *low voltage* is noted in the presence of *other* criteria for RVH, this finding *adds support* to the diagnosis of probable *pulmonary* disease (*especially in a patient with a history of smoking*).

08.27 – *Schamroth’s Sign for RVH: A Null Vector in Lead I*

Beyond-the-Core: We draw attention to an interesting, uncommon finding known as **Schamroth’s Sign**. This is the presence of a **null vector** (or very small QRS complex) in **lead I** that is not due to technical error/lead misplacement (**Figure 08.27-1**). When seen in the “right” clinical setting (ie, a patient with COPD/pulmonary disease) — then this ECG finding is *highly* suggestive of **advanced pulmonary disease** with **probable RVH** (especially if other findings in **Figure 08.24-1** are also present).

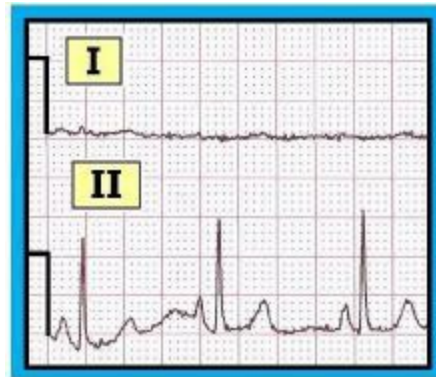


Figure 08.27-1: Schamroth’s sign. Note the “**null vector**” in lead **I**. RAA is also present (*tall, peaked P wave in lead II*). When seen in a patient with pulmonary disease and not due to technical error — Schamroth’s sign suggests disease is severe and that there is *probable* RVH (See also Section 08.33).

08.28 – RVH: *Tall R Wave in V1; RV “Strain”*

The 2 findings in **Figure 08.24-1** that best *distinguish* between pulmonary disease with vs without associated RVH are: **i)** presence of a *tall* R wave in lead V1; and **ii)** presence of RV “strain”.

- **Tall R Wave in Lead V1** — Lead V1 is a *right-sided* lead. As a result, the QRS is normally *negative* in lead V1 (*electrical activity moves toward the larger LV and away from V1*). IF the **R wave** is *taller* than the **S wave** in lead V1 — this means **rightward forces** are **increased** (*which may be an important sign of RVH*). Clinically — by the time a *tall* R wave is seen in lead V1 in an adult with pulmonary disease — the extent of **RVH** is usually **marked** (ie, *the patient is likely to have end-stage COPD and/or pulmonary hypertension*). This is the case in schematic **Figure 08.29-1** shown below.
- **RV “Strain”** — Just as LV “strain” is a sign of *true* LVH — seeing “**strain**” in one or both of the **right-sided lead areas** (**II, III, aVF** — and/or — **V1, V2, V3**) strongly **supports** a diagnosis of **RVH** (**Figure 08.29-1**).

08.29 – Schematic **FIGURE 08.29-1: Example of RVH + RV “Strain”**

Most of the ECG criteria for **RVH** are present in *schematic* **Figure 08.29-1**. Note the following:

- **Marked RAD** (*negative net deflection in lead I; positive QRS in lead aVF*).
- **RAA** (*tall, peaked P wave that looks “uncomfortable to sit on” in II, III, aVF*).
- **Tall R Wave** in lead **V1** (Section 08.28).

- **Persistence** of deep S waves in V5,V6 (Section 08.26).
- Finally — there is "**RV strain**". Typically — RV “strain” is seen in inferior and/or anterior leads (both of which are present here). **PEARL:** Inferior and/or anterior ST-T wave changes as seen in Figure 08.29-1 will often not be due to ischemia — *but rather to RVH or pulmonary embolus*. The presence of RAD; RAA; *persistent* precordial S waves; and tall R wave in lead V1 all suggest that the inferior and anterior ST-T wave changes seen in Fig. 08.29-1 are the result of pulmonary disease.

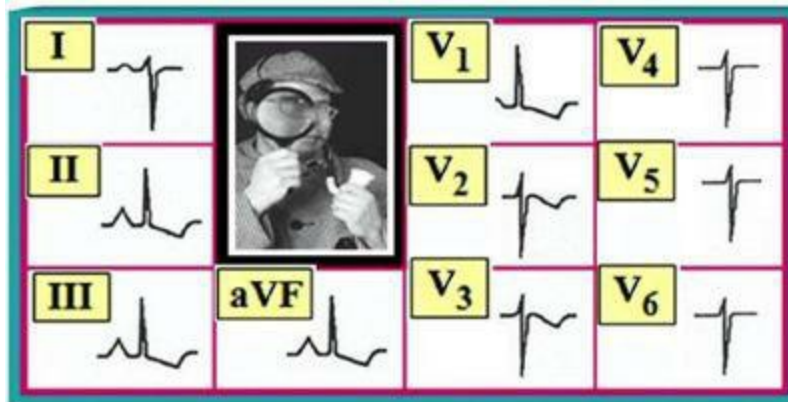


Figure 08.29-1: Schematic example of RVH + RV “strain”. This ECG picture with virtually *all* criteria from Figure 08.24-1 is *uncommon* in adults — and usually indicates *significant* RVH/*end-stage* COPD *and/or* pulmonary hypertension.

08.30 – Schematic FIGURE 08.30-1: Example of “Pulmonary” Disease

Pulmonary disease (*such as COPD*) without frank RVH may sometimes be suggested by ECG. Specifically — we look for the presence of *at least* 2 of the first 5 findings from Figure 08.24-1. This concept is illustrated in *schematic* Figure 08.30-1. Note the following:

- An *indeterminate* axis (negative QRS deflection in both leads I and aVF).
- RAA (tall, peaked P wave that looks “uncomfortable to sit on” in II,III,aVF).
- rSr’ in lead V1 (Section 08.26).
- **Persistence** of deep S waves throughout **all precordial leads** (Section 08.26). Transition *never* occurs (*the R wave never becomes taller than the S wave in the precordial leads*).

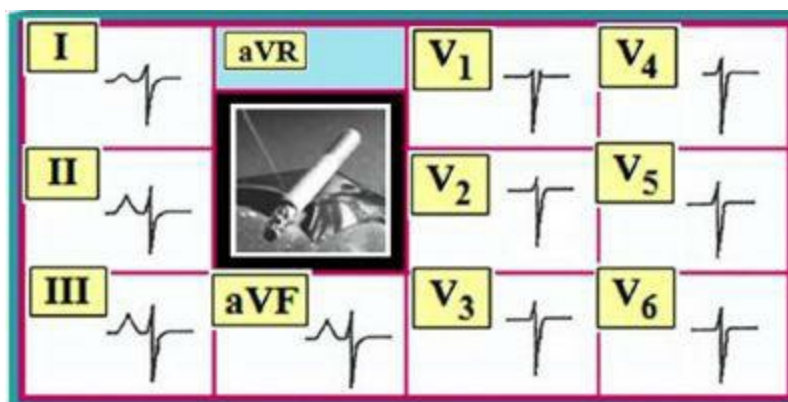


Figure 08.30-1: Schematic ECG that *strongly* suggests “pulmonary” disease. Low voltage is *not* seen

— but the other 4 criteria from Figure 08.24-1 are seen (Note — *the axis is indeterminate; there is RAA; an rSr' in $V1$; and persistence of precordial S waves*).

Clinical Note: There is no definitive ECG sign that “proves” a patient has “pulmonary disease”. Instead — We use this descriptor as a *qualitative* term that may help to explain a *combination* of ECG findings that are likely to be seen in a group of patients with longstanding/severe pulmonary disorders. As stated — the point at which “pulmonary disease” *crosses over* to cor pulmonale with *frank* RVH is often elusive and *not* detectable by ECG. An Echo would be far more informative regarding true *right-sided* chamber size, function and pressures.

- We suspect “***pulmonary disease***” when a longtime smoker presents with an ECG manifesting *at least* 2 or 3 of the first 5 findings in Figure 08.24-1.
- Given the presence of 4 of these first 5 findings in *schematic* Figure 08.30-1 — it is certainly possible that such a patient might have **frank RVH** that simply is not yet manifesting a *predominant* R wave in lead $V1$. The ECG is simply *not* sensitive enough to reliably pick up all cases of RVH.
- Remember that the adult LV is about 3 times as thick as the RV. In 3 dimensions — LV mass may normally be up to 10 times greater than normal RV mass. Development of RVH in an adult to the point that it *becomes* evident on ECG (*by a predominant R wave in lead $V1$ — as in Figure 08.29-1*) — is therefore a *late* sign.

08.31 – Pediatric RVH: A few Brief Thoughts ...

Beyond-the-Core: Interpretation of *pediatric* 12-lead ECGs is a specialty unto itself (*well beyond the scope of this ePub*). Most providers are only infrequently called upon to interpret pediatric ECGs. We therefore limit our comments here to discussion of a few basic concepts:

- ECG criteria for determining *atrial* abnormality (*RAA, LAA*) in children are similar to criteria that are used in adults (*Sections 08.19, 08.20*).
- Very *different* criteria must be used for determining ventricular enlargement in pediatric patients. Depending on the *age* of the child being assessed — voltage criteria will vary greatly.
- Detailed tables of pediatric norms are found in textbooks on this subject. Suffice it to say that QRS amplitude is often normally increased in the pediatric patient. Technical problems relating to accuracy of chest lead placement on the tiny chest of a small child when ECGs are recorded by nonpediatric providers are common. Determining whether QRS amplitude exceeds limits for age and satisfies pediatric ECG criteria for LVH is far from a simple matter. **Practically Speaking** — the child with symptoms *and/or* a murmur will be referred if there is any suspicion of chamber enlargement.

KEY Point: As opposed to the difficulty for the *nonpediatric* provider in determining LVH — ***suspected* RVH** is often far easier to surmise on ECG. In contrast to the situation just described for adults (*in which LV mass is normally up to 10 times greater than RV mass*) — RV mass is *comparable* to LV mass at birth, and tends to *remain* so for the first few years of life. As a result — a proportionately *smaller* increase in RV size is much more likely in children to produce a noticeable

change in *net* electrical activity between the two ventricles. RVH is therefore much *easier* to diagnose in young children than it is in adults. Be aware of the following:

- Some degree of **RAD** is common in young children.
- The **R wave** may normally be *taller* than the S wave is deep in **lead V1** up to *about age 5*. By the time a child is 6 years old — the S wave in lead V1 will usually be deeper than the R wave in this lead is tall.
- It is common to see *anterior* T wave inversion on the ECG of otherwise healthy children. This ECG phenomenon is known as a **juvenile T wave variant**. Symmetric T wave inversion may extend to as far as lead V3 or V4 as a *normal* phenomenon in a young child or early adolescent. *Clinical correlation* is essential to avoid mistaking this normal T wave variant as a pathologic pattern (*Section 01.4*).
- **IE** in doubt about “norms” — Refer to *age-specific* tables! Have a low threshold for consulting with someone having expertise in pediatric ECG interpretation.

Pediatric ECGs: Is RVH Likely?

Imagine the *schematic* ECGs shown in **Figure 08.31-1** and **Figure 08.31-2** were each obtained from a 2-year old child. *Is RVH likely?*

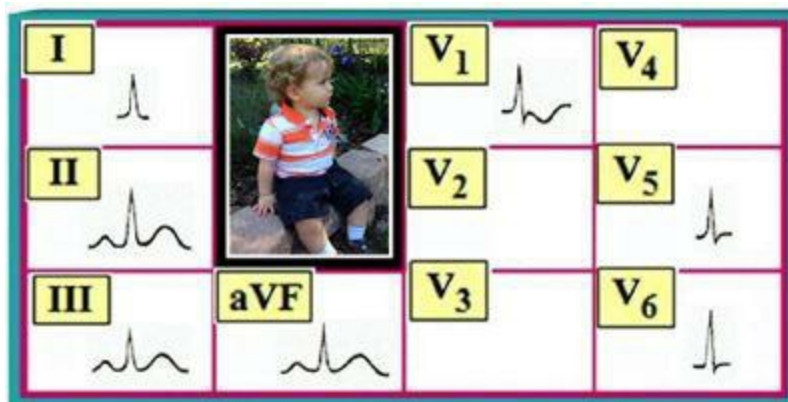


Figure 08.31-1: *Schematic* ECG from a young child. Although there is a *predominant* R wave in lead V1 — this may normally be seen up to 5 years of age. Lack of other findings suggests this 2 year old probably does not have RVH.

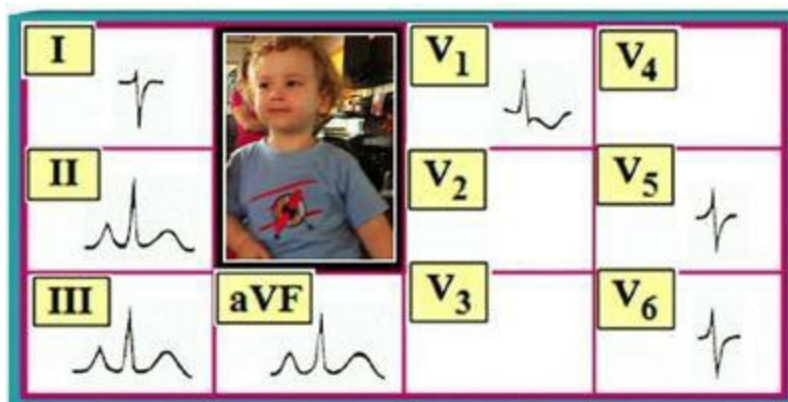


Figure 08.31-2: *Schematic* ECG from a young child. RVH is suggested by a *combination* of findings including RAD; RAA; *tall* R wave in lead V1 *with* RV “strain”; and persistent deep S waves in V5,V6 (*See text*).

08.32 – FIGURE 08.32-1: *Is there RVH?*

The ECG in Figure 08.32-1 was obtained from an older adult with *new-onset* shortness of breath *thought* to be due to *congestive* heart failure.

- *What else* do you suspect?

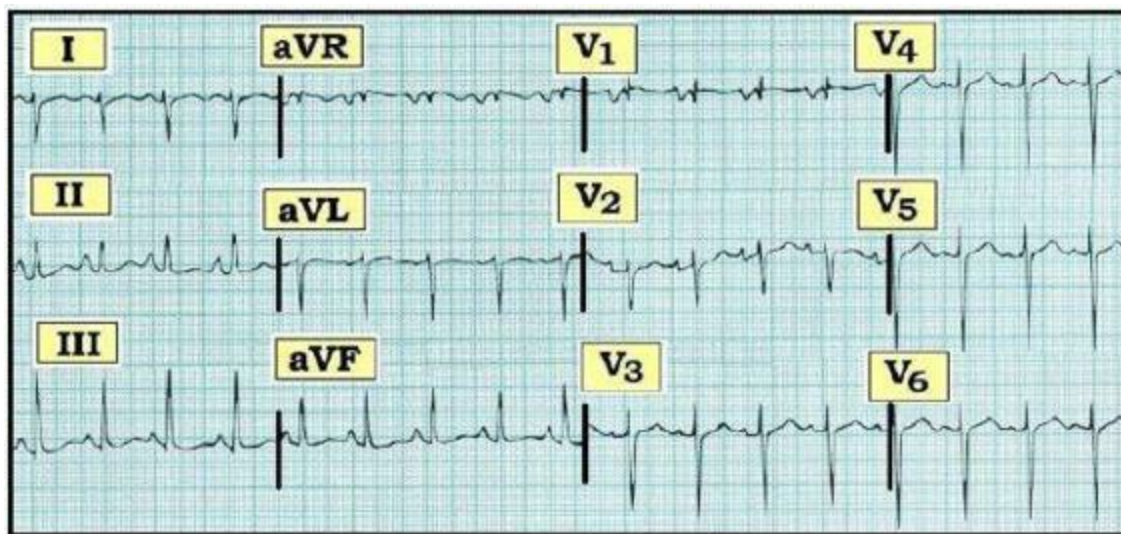


Figure 08.32-1: ECG from a patient with heart failure. Is this ECG what you would expect from a patient with *left-sided* heart failure? (See text).

Answer to Figure 08.32-1: The rhythm is sinus tachycardia at ~115/minute. The QRS is narrow. As opposed to the usual picture of *left-sided* heart failure (*in which one expects to see LVH with strain*) — there is *no* sign on this ECG of LVH. Instead — the tracing strongly suggests **severe pulmonary disease** (*if not frank RVH*):

- There is **marked RAD** (*predominantly negative QRS complex in lead I; positive QRS in lead aVF*).
- **RAA** is present (*tall, peaked, “uncomfortable-to-sit-on” P wave in inferior leads that in lead II is clearly >2.5mm*).
- An **incomplete RBBB** is seen (*rsR’ in lead V1; S waves in leads I, V6*). Note that the r’ in lead V1 is relatively tall.
- **Deep S waves** persist through to V5, V6.
- There are *nonspecific* ST-T wave abnormalities — albeit *not quite* RV strain (*and no acute changes*).

Bottom Line: ECG Impression — The combination of *marked* RAD, *definite* RAA, IRBBB and *persistent* precordial S waves *all* point to a diagnosis of **RVH** for the ECG shown in Figure 08.32-1.

- **Clinical Impression:** This ECG should make one *rethink* the premise of *left-sided* heart failure as the primary cause of this patient’s *new-onset* shortness of breath. Instead — longstanding/severe **pulmonary disease** is likely given the **combination** of ECG findings.

Depending on the history (*and comparison with prior ECGs on this patient*) — **acute pulmonary embolism** might also be considered as a possible diagnosis (See Section 08.34).

- *Beyond-the-Core*: There is also **LAA** in [Figure 08.32-1](#) (*very deep negative component to the P wave in lead VI*). Whether this reflects anatomic enlargement vs increased LA pressure is uncertain.

08.33 – FIGURE 08.33-1: *Is there RVH?*

We conclude this section on RVH with the ECG shown in [Figure 08.33-1](#). We have previously seen this tracing (*in* [Figure 02.17-1](#)).

- Does this ECG suggest *pulmonary* disease?
- Is there RVH?

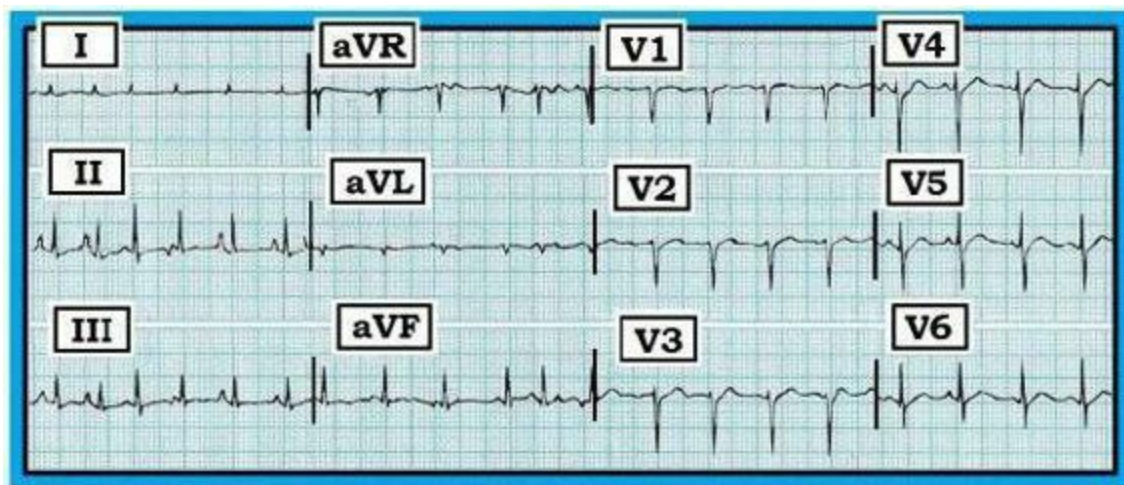


Figure 08.33-1: ECG reproduced from [Figure 02.17-1](#). Is there evidence of *pulmonary* disease? Is RVH likely?

Answer to Figure 08.33-1: This patient *undoubtedly* has severe pulmonary disease (*if not frank RVH*). We note the following findings:

- The rhythm is **MAT** (Sections 02.16, 02.17) — as determined by the *irregular* irregularity with multiple *different* P wave morphologies that *change* from beat-to-beat. The presence of MAT is most often associated with *severe* pulmonary disease.
- Several of the P wave forms that are seen in lead II are highly suggestive of **RAA** (*tall, peaked and pointed P waves in this lead*).
- There is *persistence* of fairly **deep S waves** in *lateral* precordial leads.
- **Schamroth's Sign** is present — as suggested by the *very* low amplitude r wave with flat ST-T wave in lead I (Section 08.27).

Clinical Impression: The ECG in [Figure 08.33-1](#) was obtained from a patient known to have COPD. The *combination* of ECG findings noted above should provide insight that the degree of pulmonary

disease is severe, if not already associated with RVH and increased pulmonary pressures.

08.34 – Acute *Pulmonary Embolus*



The ECG is usually *not* a good tool for diagnosing **acute pulmonary embolism** (*acute PE*). That said — there *are* times when an ECG may strongly suggest the diagnosis *before* V/Q scan or chest CT can be done. **Acute PE** should be considered IF:

- IF the **clinical setting** is “**right**” (*new-onset dyspnea — pleuritic chest pain — predisposing risk factors or previous history of PE or deep venous thrombosis*).
- IF the patient has **sinus tachycardia** (*usually seen with large PE, albeit clearly nonspecific for the diagnosis*).
- Two or more ECG signs of **acute “right-heart” strain**. These include: **i)** RAD; **ii)** RAA; **iii)** IRBBB or RBBB; **iv)** Tall R wave in lead V1; and **v)** Deep S waves in leads V5,V6 (*Section 08.24*).
- **RV “strain”** — as suggested by *asymmetric ST-T wave depression in inferior and/or anterior leads (ie, leads II,III,aVF and/or V1,V2,V3)*. Alternatively — there may only be **nonspecific ST-T wave changes** (*ST flattening; slight ST depression*).
- **New-onset A Fib** (*a common but nonspecific finding with acute PE*).
- History and the ECG are similar to that presented in Section 08.32.
- Possibly with an **S1-Q3-T3 pattern** ... (*Section 08.36*).

08.35 – Acute PE: Key Clinical Points

From a clinical perspective — *Acute PE* is **increasingly overdiagnosed!** Many small (*subsegmental*) PEs *unlikely* to be of clinical consequence are being identified by **overuse of Chest CT** ordered on *lower-risk* patients with atypical symptoms.

- The ECG is **unlikely** to **identify** patients with **smaller PEs**. *Don’t expect to see anything on the ECG of these patients (this may be a blessing in disguise)*.
- **Overall** sensitivity of the ECG for picking up *acute PE* is *poor*. Failure to see any of the ECG signs in Section 08.34 does *not* rule out the possibility of acute PE IF history and clinical features suggest the diagnosis. *Chest CT is needed*.
- **Sensitivity** of the ECG for suggesting the possibility of acute PE is *better* with larger (*especially submassive*) PEs. One should *at the least* see **sinus tachycardia** in such patients — often with **nonspecific ST-T wave changes**. Large acute PE is commonly associated with other ECG signs — but these may be subtle and are generally nondiagnostic.
- Be aware of the *clinical* significance of **RV “strain”**. The finding of **anterior T wave inversion** (*in leads V1,V2,V3*) in an adult with *new-onset dyspnea or pleuritic (or atypical) chest pain* will much more often be due to **acute PE** than to ischemic heart disease. This is especially true IF there is also *inferior T wave inversion (in II,III,aVF)* — as seen in **Figure 08.35-1**.

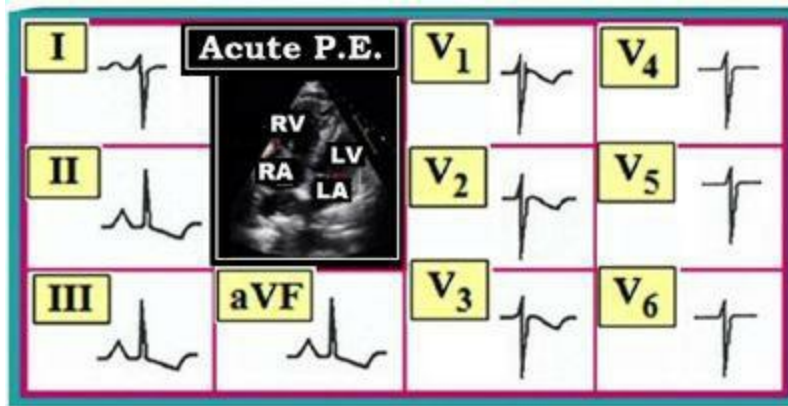


Figure 08.35-1: Schematic ECG illustrating a series of ECG findings that taken together are *highly* suggestive of **acute PE** (*Pulmonary Embolism*) — especially when seen in a patient with *new-onset* dyspnea. Note **RV “strain”** — in the form of *asymmetric* ST-T wave depression seen here in both *inferior* and *anterior* leads (*II,III,aVF*; and *V1,V2,V3*). In addition — there is **RAD** (*the QRS in lead I is markedly negative*) — **RAA** (*tall, peaked P wave in inferior leads*) — **incomplete RBBB** (*rSr’ in V1; S waves in I,V6*) — and **persistent precordial S waves** that remain *deep* up to leads V5,V6.

08.36 – FIGURE 08.36-1: Should You Look for an S1-Q3-T3?

Over the years — much attention has been focused on the diagnostic utility of recognizing an **S1-Q3-T3 pattern** as an ECG indicator of **acute PE**. While important to be aware of what a “*positive S1-Q3-T3 pattern*” is — We feel this ECG sign is *highly* overrated. Our reasons for stating this are given below.

What is a Positive S1-Q3-T3 Sign?

We define an S1-Q3-T3 pattern — as the presence of: **i)** an S wave in lead I; **ii)** a Q wave in lead III; and **iii)** T wave inversion in lead III (**Figure 08.36-1**):

- In order to be valid — all 3 of these ECG indicators must be present! That is — **lead III must** show both a **Q wave** and an **inverted T wave**! The presence of only 2 ECG indicators does not qualify as a positive S1-Q3-T3 pattern.

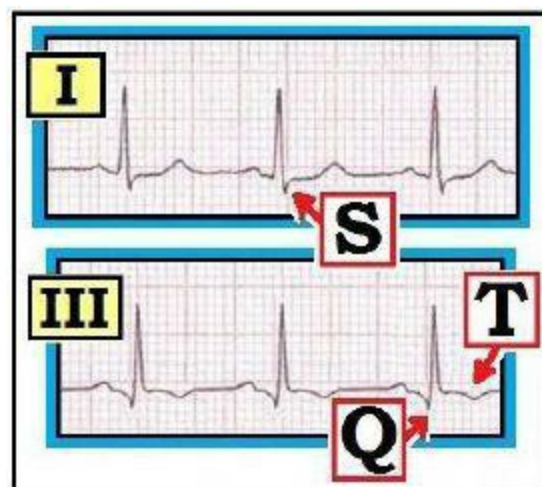


Figure 08.36-1: S1-Q3-T3 Pattern — as recognized by the presence of: **i)** an S wave in lead I; **ii)** a Q wave in lead III; **and iii)** T wave inversion in lead III. To be valid — all 3 of these ECG indicators must be present.

The S1-Q3-T3 Pattern: Pros and Cons

By itself — one can not rely on a *positive* S1-Q3-T3 sign as an ECG indicator of acute PE. This is because even in the *best* of studies — both sensitivity and specificity of a *positive* S1-Q3-T3 sign for *acute* PE is poor. That is — Failure to see this sign *in NO way* rules out the possibility of *acute* PE.

- More often than not — you will not see an S1-Q3-T3 sign despite even large *acute* PE.
- We have seen the S1-Q3-T3 sign *many times* in completely *healthy* and *asymptomatic* adults! Clearly — these patients do not have *acute* PE.
- **BOTTOM Line:** Recognition of a *positive* **S1-Q3-T3 pattern** *may* help in the diagnosis of *acute* PE — IF the *positive* S1-Q3-T3 pattern is present in association with other suggestive ECG signs and the “*right*” clinical history. But a positive S1-Q3-T3 is not helpful by itself — and absence of a positive S1-Q3-T3 on ECG *rules out nothing*.

08.37 – FIGURE 08.37-1: The Cause of Anterior T Inversion?

Imagine the ECG in Figure 08.37-1 was obtained from an adult with *new-onset* atypical chest discomfort and shortness of breath.

- Given this clinical scenario — *How would you interpret the **symmetric** T wave inversion* seen in leads V1, V2, V3?

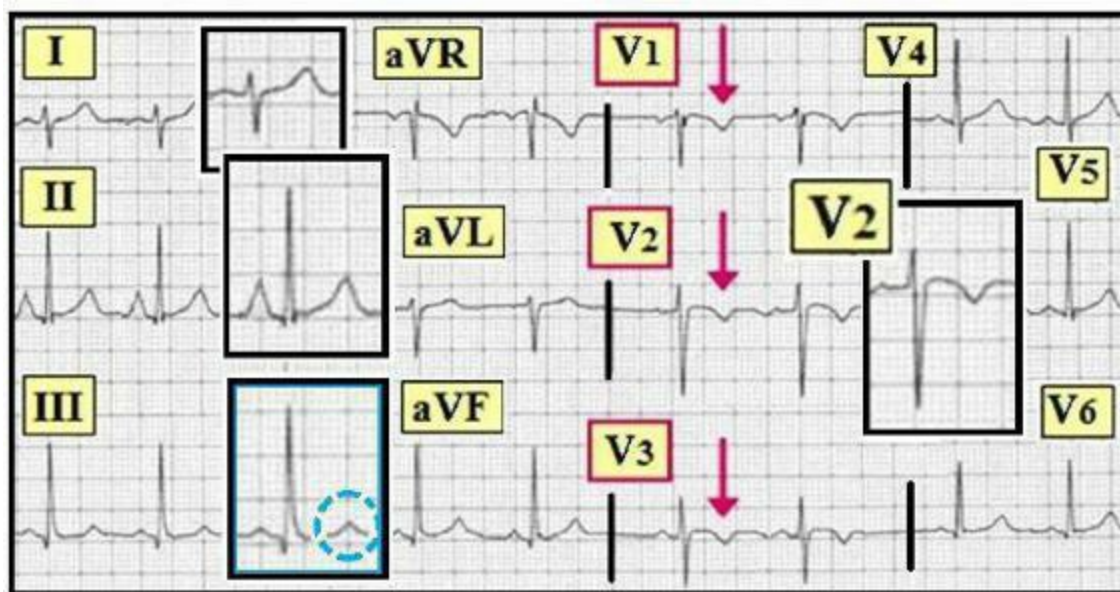


Figure 08.37-1: ECG obtained from a patient with atypical chest pain and *new-onset* dyspnea. How would you interpret the *anterior* T wave inversion (*arrows*) seen here? Note that an S1-Q3-T3 pattern is not present (*there is an S1-Q3 but no T3 as highlighted by the blue-white circle*).

Answer to Figure 08.37-1: The rhythm is sinus. There is **RAD** (*the QRS complex in lead I is*

predominantly negative) — **RAA** (*tall, peaked and pointed P wave in lead II*) — and fairly **deep, symmetric T wave inversion** is seen in **leads V1,V2,V3** (*arrows in Figure 08.37-1*).

- The finding of *symmetric T wave inversion* in a patient with chest discomfort should always prompt consideration of **ischemia** (*which we would do in this case*). That said — one should especially consider acute PE in this case because: **i)** The patient also has *new-onset* dyspnea; and ii) there are *other* signs suggestive of *acute* “RV strain” on this tracing (*marked RAD; RAA*). As a result — **Acute PE** would be our **1st diagnosis!**
- **NOTE:** An **S1-Q3-T3** is not present in Figure 08.37-1 — because the T wave is not inverted in lead III (*blue-white circle seen in the lead III blow-up*). That said — *lack of an S1-Q3-T3* pattern should never be used alone to rule in or out acute PE. While possible ischemia must be considered — the history of *new-onset* dyspnea and this ECG showing *anterior* T wave inversion plus marked RAD and RAA makes *acute* PE our working diagnosis until proven otherwise.

08.38 – FIGURE 08.38-1: *Is there Acute Anterior STEMI?*

The ECG in Figure 08.38-1 was obtained from a patient who presented with syncope, shock, and acute hypoxemia. In addition to **RBBB** (*QRS widening with an rSR' in V1 and S waves in I, V6*) — there is ST coving in V1,V2,V3 and *slight-but-definite ST elevation* in **leads V1,V2**.

- In view of this clinical scenario — Is this ECG suggestive of **acute STEMI** (*ST Elevation Myocardial Infarction*)?
- Should you activate the cath lab based on this tracing?

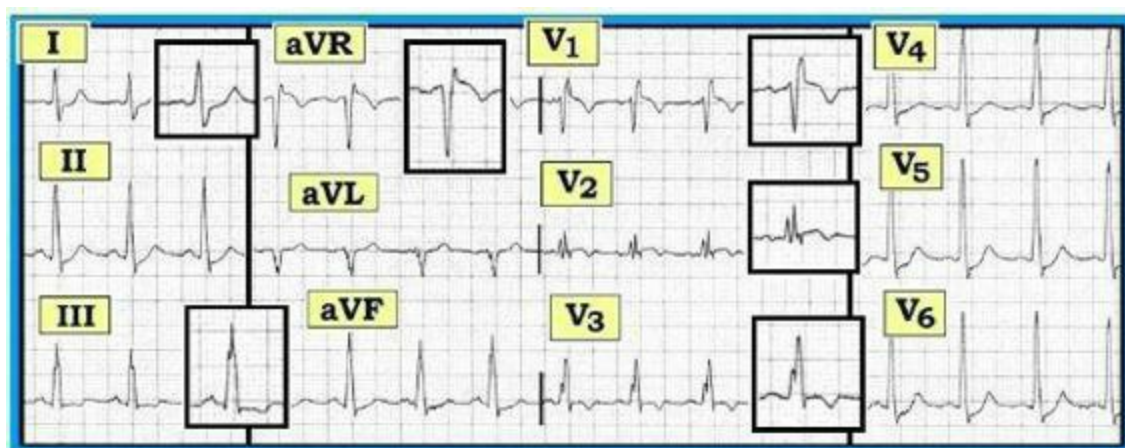


Figure 08.38-1: ECG obtained from a patient who presented with syncope, shock and acute hypoxemia. Is this ECG suggestive of acute *anterior* STEMI?

Answer to Figure 08.38-1: This ECG needs to be interpreted as the “sum” of its parts *in context* with the clinical history. Remarkably *absent* from the history in this patient who presented with syncope, shock and acute hypoxemia — is any mention of chest pain. ECG findings in **Figure 08.38-1** include:

- **Sinus tachycardia** (rate ~ 100/minute).
- **RBBB** (*QRS widening to at least 0.11 second; rSR' in lead V1; wide S in lead I; subtle-but-present s wave seen in lead V6*).
- **Diffuse ST-T wave changes** (See below).
- Presence of an **S1-Q3-T3** pattern as a *supportive* ECG sign (**Figure 08.38-2**). Although the q wave and T inversion in lead III are admittedly subtle — *each* component of the S1-Q3-T3 pattern is present (red arrows in Fig. 08.38-2).

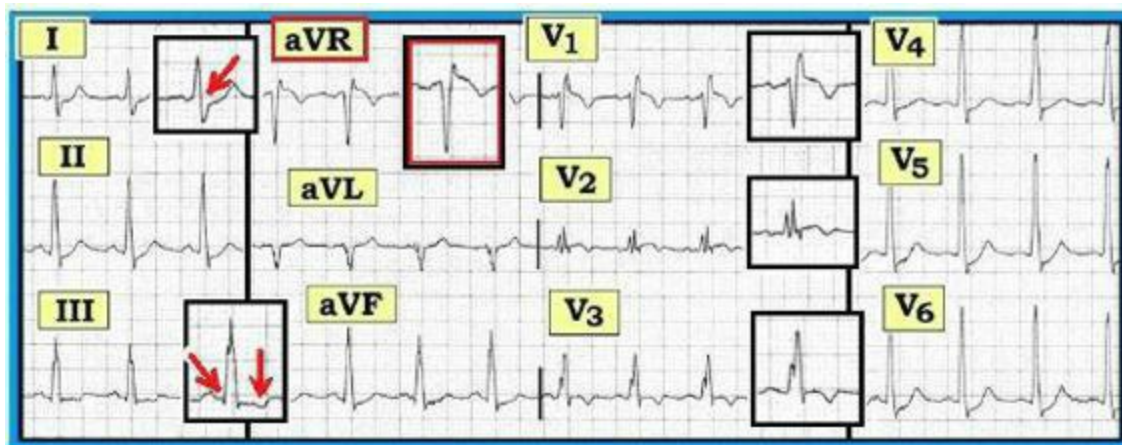


Figure 08.38-2: We have added *red* arrows to Fig. 08.38-1 to highlight the presence of an **S1-Q3-T3** pattern. Other ECG findings suggestive of **acute PE** given the history include: **i)** Sinus tachycardia; **ii)** RBBB; **iii)** *diffuse* ST-T wave changes; and **iv)** ST elevation in lead aVR (See text).

Clinical Impression of Figure 08.38-2: We emphasize — that one can *not* rule out the possibility of acute *anterior* STEMI from the ECG shown in Figure 08.32-2. That said — virtually *all* facets of the history and this ECG can be explained by one *unifying* diagnosis = **acute PE**:

- Patients with *submassive* acute PE commonly present with syncope, shock *and/or* acute hypoxemia. IF these symptoms were the result of *acute* STEMI — We would expect far more extensive ST elevation than is seen in Figure 08.38-2.
- In the overall clinical context of this case — the **symmetric anterior T wave inversion** with **diffuse ST depression** elsewhere (in *inferior and lateral precordial leads*) is perfectly consistent with **acute RV “strain”**.
- As expected — **sinus tachycardia** is present.
- **RBBB** is another common sign of acute RV “strain”.
- A *limited* number of conditions produce **ST elevation** that is most **marked** in lead **aVR** — but minimal or *nonexistent* elsewhere, with possible exception of lead V1 (See Sections 09.31-through-09.40). One of these conditions is **acute PE** (Section 09.33). Note that there is **marked** ST elevation in lead aVR of Figure 08.32-2 (red blow-up box) — with ST elevation otherwise limited to lead V1 and no more than a trace in lead V2.
- **BOTTOM Line:** Putting all clues together — **acute PE** should be assumed until proven otherwise. This patient was found to have a large saddle embolus.

➡ Q-R-S-T Changes

09.1 – FIGURE 09.1-1: *Assessing Q-R-S-T Changes*

The "heart" of ECG interpretation resides with assessing the tracing for **QRST Changes**. The purpose of this mnemonic — **Q-R-S-T** — is to ensure a **systematic approach** — *so that nothing is left out*. The most common mistake that occurs when a systematic approach is *not* closely followed is to allow more dramatic ST-T wave changes to consume one's attention — while subtler (*but equally important*) findings go unnoticed. Examples of such *all-too-easy-to-overlook* findings include **failure to recognize non-sinus** rhythms; a *dominant* R wave in lead V₁; *poor* R wave progression and subtle ST-T wave changes.

- **BOTTOM Line:** Always use a **Systematic Approach**. This *not only* prevents overlooking important ECG findings — but also **saves time** by *streamlining* and *organizing* your approach (**Figure 09.1-1**):

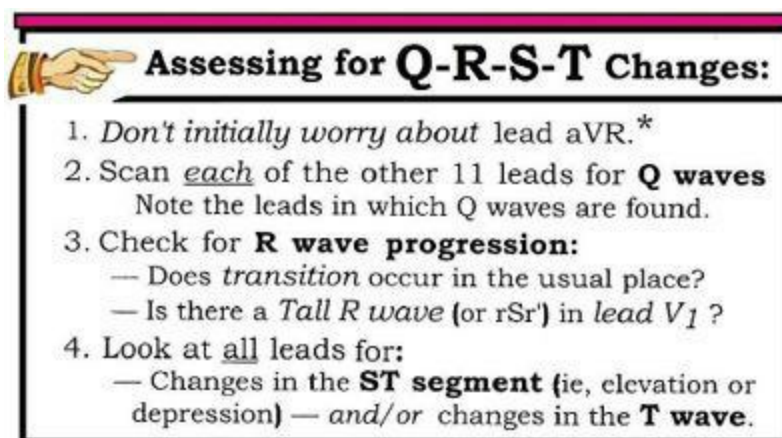


Figure 09.1-1: Systematic approach for assessing Q-R-S-T Changes (See text).

LEAD aVR: Largely ignored in the past — Use of lead aVR is now increasingly appreciated as an **invaluable adjunct** to ECG interpretation. We fully explore this *advanced* concept in Sections 09.31-through-09.40. In the meantime — We suggest *not* being overly concerned about the ECG appearance in lead aVR:

- For the most part — assessment of lead aVR is *not* essential to the basics of ECG interpretation (*assuming the leads are on correctly and there is no dextrocardia*).
- **Beyond-the-Core:** ECG appearance in **lead aVR** may assist in assessing not only technical errors — but *also* narrow and wide-complex tachycardias; acute coronary syndrome from multivessel or left main disease; pericarditis; pulmonary embolism; and more (*See Sections 09.31-through-09.40*).

09.2 – Septal Depolarization: Reason for Normal Septal Q Waves

In Section 05.7 — We introduced the concept of **normal septal activation**. Normally — the very first part of the ventricles to depolarize (*after the impulse passes through the AV node and Bundle of His*) — is the **left side** of the **ventricular septum**. As a result — septal depolarization **normally** moves from **left-to-right** (red arrow pointing left-to-right in **Panel A** of Figure 09.2-1).

- As a result of this initial *left-to-right* direction of *septal* depolarization — **left-sided leads** (*I, aVL; V4, V5, V6*) normally see *initial* ventricular activation as moving *away* — which is why **small septal q waves** are often *normally* seen in one or more of the *lateral leads* (**Panel A**).
- In contrast — **right-sided leads** (*V1, V2*) normally view *initial* ventricular activation as coming *toward* the site on the chest where these leads lie. This explains why a **small initial r wave** is commonly seen in **leads V1, V2** (See lead *V1* in **Panel A**). This initial small r wave represents *septal* depolarization. It is *lost* when there is *septal* infarction (See Figure 09.3-1 — in Section 09.3).

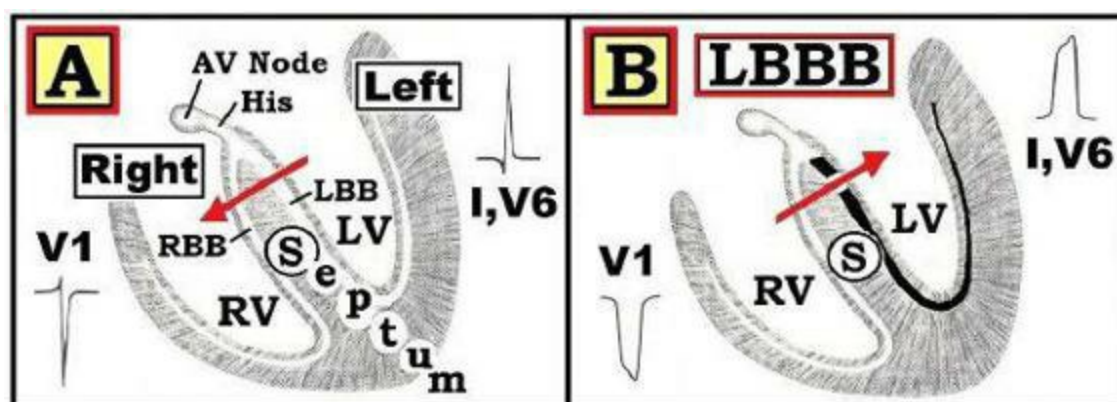


Figure 09.2-1: Normal *septal* activation (A) vs LBBB (B). Septal activation normally begins on the *left* side of the septum — and travels *left-to-right* (red arrow in **Panel A**). As a result — one normally sees a small r wave in right-sided leads (*V1, V2*) and small, narrow q waves in one or more of the lateral leads (*I, aVL; V4, V5, V6*). **RBBB** does not change these findings (*since the right bundle branch travels down the right side of the septum*). In contrast — **LBBB** reverses the direction of septal activation (red arrow in **Panel B**). As a result — one should *never* normally see a septal q wave in a *lateral* lead with LBBB, unless there has been *septal* infarction (See text).

What Happens with BBB?

Since the **RBB** (*Right Bundle Branch*) travels down the *right* side of the septum — the initial **left-to-right** direction of *normal* septal depolarization does not change when there is **RBBB** (Section 05.7).

- In contrast — with **LBBB**, the normal *left-to-right* direction of septal activation will *reverse* because the block with LBBB prevents the initial vector of septal activation from starting on the left (**Panel B** in Figure 09.2-1). As a result — ventricular activation with **LBBB** moves almost entirely from **right-to-left**. This explains why septal q waves should not normally be seen with LBBB in *lateral* leads (Sections 05.6-through-05.9).

09.3 – *Precordial Lead Appearance: What is Normal?*

In **Figure 09.3-1** — We illustrate normal *precordial* lead appearance by means of a *schematic* cross-sectional (*transverse*) view of the heart within the thorax:

- The **small black arrow** in **Figure 09.3-1** schematically depicts the direction of normal septal depolarization (*which moves from left-to-right*).
- The **large red arrow** depicts the general direction of LV (*Left Ventricular*) depolarization (*which as seen in Fig. 09.3-1 is to the left and posteriorly*).
- The smaller **RV** (*Right Ventricle*) — predominantly sees electrical activity as moving *away* from the right (*and toward the larger and thicker left ventricle*). This explains why the QRS complex in **right-sided precordial leads** (*V1,V2*) is normally predominantly *negative* — whereas the QRS in **left-sided leads** (*V5,V6*) is generally positive.

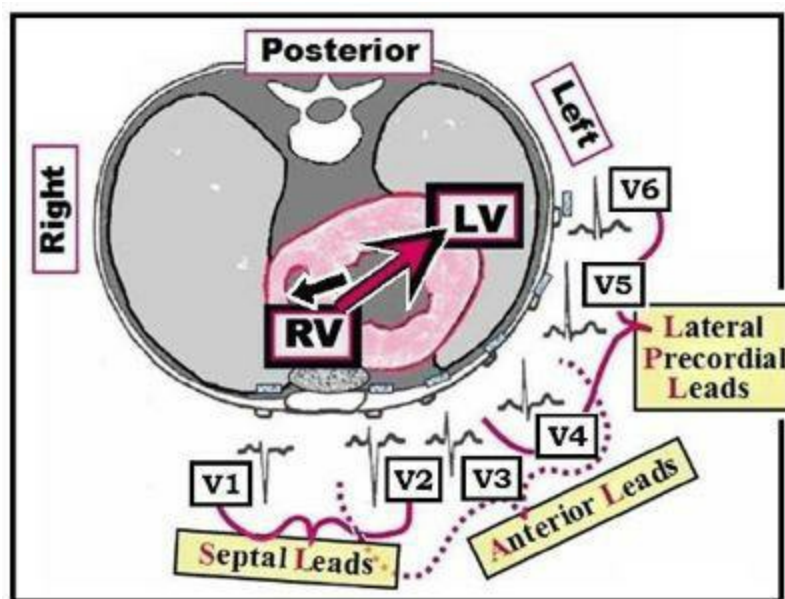


Figure 09.3-1: Transverse (*cross-sectional*) view of the heart — illustrating *precordial* lead appearance in leads V1-through-V6. Note that transition occurs *between* lead V2-to-V4. There is **overlap** between leads viewing *septal* — *anterior* — and *lateral* precordial areas. Septal depolarization normally moves left-to-right (*small black arrow*). The major component of ventricular activation moves to the left and posteriorly (*large red arrow*) — which reflects the relative size and anatomic position of the left ventricle.

FIGURE 09.3-1: Key Clinical Points

- Note that there is **overlap** in **Figure 09.3-1** between leads viewing *septal* — *anterior* — and *lateral* precordial lead areas. That is — **lead V2** is both a septal and anterior lead; **lead V4** is both an anterior and lateral lead.
- **Septal depolarization** (*the small black arrow in Figure 09.3-1*) goes *left-to-right*. The normal *small* initial positive deflection (*r wave*) in leads V1,V2 results from *septal* activation coming toward these *right-sided* leads. With **septal infarction** — this small *initial* r wave in leads V1,V2 is *lost*.
- Small **normal septal q waves** — are commonly seen in one (*or more*) of the **lateral leads**

(*I, aVL; V4, V5, V6*). This makes sense — since the **left-to-right** direction of **septal activation** moves away from *left-sided* leads (and therefore writes a **small q wave**).

- The area where the R wave *becomes* taller than the S wave (**Transition**) occurs normally in Figure 09.3-1. That is — the QRS is predominantly *negative* in lead V2 — it is *isoelectric* in V3 — and positive in V4 (so **transition** occurs between leads V2-to-V4, which is normal).

09.4 – Basic Lead Groups: Which Leads look Where?

It is essential to know *which* leads view *which* areas of the heart. The “**trained eye**” keys into specific **Lead Groups** (Figure 09.4-1):

- In Sections 03 and 07 — we introduced derivation and anatomic correlation for the 6 limb leads in the frontal plane. This includes the 3 **inferior leads** (*II, III, aVF*) — the 2 **high-lateral leads** (*I, aVL*) — and the most remote (*right-sided*) lead, which is lead **aVR**. We expand on these relationships in Figure 09.4-1.
- Think of the *electrical* viewpoint of lead **aVL** as *looking down* at the heart from perspective of the *left* shoulder. Thus, lead aVL views the heart’s electrical activity from *higher* perspective than *precordial* leads that are placed on the chest. **Bipolar lead I** at *horizontal* perspective (corresponding to 0 degree placement in Einthoven’s triangle) — also views the heart’s electrical activity from *higher* perspective than is seen from precordial leads. This is why **leads I and aVL** are designated “**high lateral**” leads — to be distinguished from *lower lying* lateral precordial leads (*V4, V5, V6*).
- In contrast — Think of the *electrical* viewpoint of lead **aVF** as looking *straight up* from the **Feet** (*perpendicular in the frontal plane axis at +90 degrees*). The other 2 **inferior leads** are lead **II** (at +60 degrees) and lead **III** (at +120 degrees).
- The remaining 6 leads are the **precordial leads**. Anatomic localization of these 6 chest leads is best understood by reviewing the landmarks used for *performing* an ECG (*Section 03.6*) in context with the *horizontal* plane view just discussed in Figure 09.3-1. This should explain **septal** (*V1, V2*) — **anterior** (*V2, V3, V4*) — and **lateral** (*V4, V5, V6*) **precordial lead** viewpoints for these *unipolar* leads that view the heart’s electrical activity from perspective of their placement site on the chest.

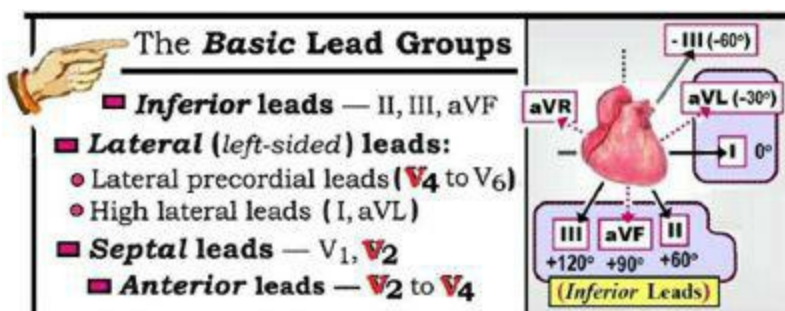


Figure 09.4-1: Basic Lead Groups. As suggested in Fig. 09.3-1 — there is **overlap** in the **precordial** lead areas (*lead V2* reflects both septal and anterior events; *lead V4* reflects both anterior and lateral events).

About Lead aVR: — The most remote of the 12 standard leads is **lead aVR**. This *right-sided* lead views the heart's electrical activity from the distant perspective of the *right* shoulder. Lead aVR is unique for providing a view that assesses the basal part of the interventricular septum. It is also unique in providing a ***mirror-image* view** of ongoing electrical activity throughout the rest of the heart.

- Note that we do *not* assign a specific number of degrees to the vantage point for **lead aVR** (Figure 09.4-1). Instead — it suffices to think of this lead as providing a remote *right-sided* view. Lead aVR may show ***reciprocal* changes** to what occurs in other areas of the heart. Diffuse ST depression in multiple leads from subendocardial ischemia in a patient with severe coronary disease — may therefore manifest ***mirror-image* ST elevation** that is only seen in **lead aVR** (This concept discussed in detail in Section 09.40).

09.5 – R Wave Progression: *Where is Transition?*

The “**R**” of our *suggested Q-R-S-T* memory aid — serves to remind us to assess *each* tracing for **R Wave Progression**. A typical sequence of R wave progression across the 6 precordial leads is illustrated in **Figure 09.5-1**, which is a *close-up* view taken from the lower part of Figure 09.3-1.

- Normally the **R wave** becomes ***progressively* taller** as one moves across the precordial leads. The R wave often (*but not always*) “**tops out**” in lead V5 — and then drops off in amplitude by lead V6. These relationships are shown in Figure 09.5-1.
- The easiest way to *quickly* describe evolution of the QRS waveform from a predominantly *negative* complex in V1,V2 — to a predominantly *positive* complex in more lateral precordial leads — is by commenting on the point of “**Transition**”, which indicates the leads *between* which the QRS becomes more positive than negative. Transition *normally* occurs between lead V2-to-V3 — or between V3-to-V4. In **Figure 09.5-1** — Transition occurs ***between* leads V2-to-V4** (since the QRS is predominantly negative in V2 — isoelectric in V3 — and clearly positive by V4).

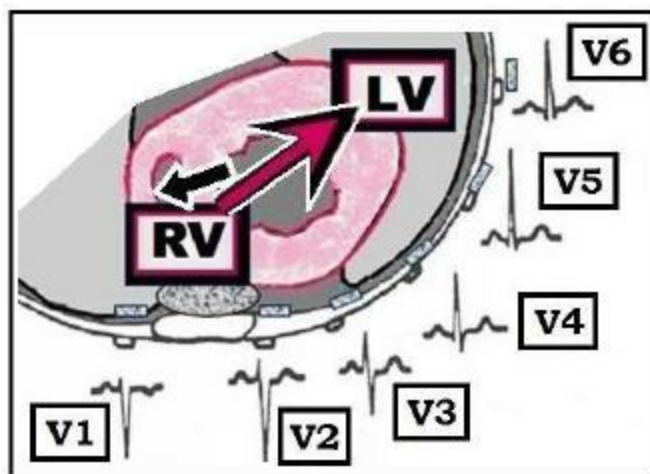


Figure 09.5-1: Close-up of the cross-sectional view shown in Figure 09.3-1 — in which *progression*

of QRS complexes in the **precordial leads** is shown. Note the *small* r wave in lead V1 and that there are small *septal* q waves in leads V5,V6. R wave amplitude “*tops out*” in V5; **Transition** is **normal** (*occurs between leads V2-to-V4*).

Where is Transition?

Determining the point of transition is easy. It is a highly *reproducible* determination — and *not* subject to *inter-interpreter* variability (**Figure 09.5-2**). In contrast — there is great variation in the way different interpreters evaluate an ECG for the adequacy of R Wave Progression (*Section 09.7*).

- **Transition** is said to be **Normal** — IF it occurs *between* leads V2-to-V4 (**Panel B** in **Figure 09.5-2**). Regardless of r wave amplitude in the first few precordial leads — the R wave becomes taller than the S wave is deep by lead V3 or V4. In **Panel B** — transition occurs *between* leads V3-to-V4.
- Transition is said to be **Early** — IF the R wave becomes taller than the S wave is deep by lead V2. This is the case in **Panel A** — in which transition occurs *between* leads V1-to-V2.
- Transition is said to be **Late** — IF the point at which the R wave becomes taller than the S wave is deep is delayed to *after* lead V4. This is the case in **Panel C** — in which transition occurs *between* leads V4-to-V5.

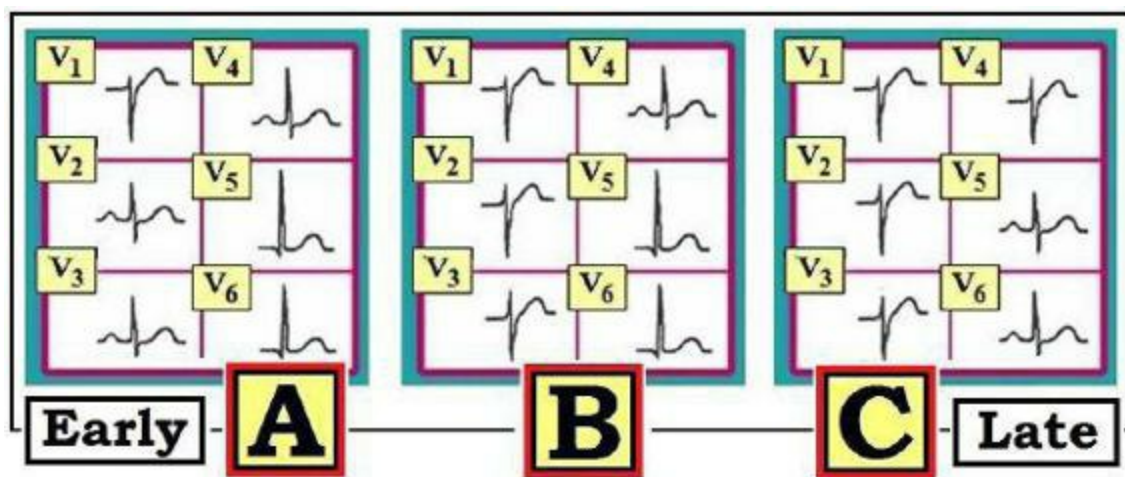


Figure 09.5-2: Transition in the precordial leads. Normal transition occurs *between* leads V2-to-V4 (**Panel B**) — vs Early (**A**) or Late (**C**) transition.

KEY Clinical Points: There is *no doubt* about where transition occurs for each of the 3 precordial lead sequences shown in **Figure 09.5-2**. Subjectivity does *not* enter into the definition.

- **NOTE:** The point of **transition** is a **descriptive finding**. The clinical significance of normal, early or late transition — will depend on *other* ECG parameters (*as will be discussed shortly*).
- Our purpose for **routinely including** assessment of **R wave progression** and the point of **transition** in our **Systematic Approach** (= the “**R**” of *Q-R-S-T Changes*) — is *not* to overlook the finding of a *disproportionately* tall R wave in lead V1. Careful assessment of precordial lead morphology should *also* pick up: **i)** any r’ that might be present in lead V1; and **ii)** any Q waves or *loss* of r wave that might occur in precordial leads (*Section 09.8*).

09.6 – Old Terminology: *R Wave Progression* – *CW, CCW Rotation*

ECG description of precordial lead QRS appearance has evolved over time. In the past — the term “*R Wave Progression*” was universally used to convey *progressive* increase (or *lack thereof*) of QRS amplitude as one *successively* views the precordial leads.

- **R Wave Progression** was said to be “*normal*” — IF the R wave gradually and *progressively* became taller as one moved across the precordial leads (*at least until leads V4, V5*).
- **PRWP** (*Poor R Wave Progression*) was said to be present — IF the R wave in lead V1 *through* to leads V3-V4 either did *not* become taller, or only increased *slowly* in size.
- Abnormalities in the way the R wave did or did not progress were described by rotation “direction”. **CCW** (*CounterClockWise*) **rotation** was said to exist in the transverse plane — IF the zone of transition occurred *before* lead V3 — vs **CW** (*ClockWise*) **rotation** IF transition occurred *after* lead V4.

Old vs New Terminology: Use of the ECG designations of “clockwise” and “counterclockwise” rotation is *outdated*. These terms are *no longer* actively used. We find these terms confusing — because they are based on the unexpected premise of an observer standing at the feet of the patient *looking upward* to envision precordial lead progression of R wave amplitude. We suggest you **ignore** the terms “*clockwise*” and “*counterclockwise*” **rotation**, which do not contribute to meaningful ECG interpretation.

- In contrast — We have kept the term “***R Wave Progression***” (*RWP*) in our ECG vocabulary, because this term remains *ingrained* in the minds of many ECG interpreters.
- **Clinical Perspective:** It is better to determine *precordial* lead “***Transition***” than to worry about R wave “progression”. The point of transition much more *reproducibly* describes what occurs in the transverse plane (*Section 09.5*).

09.7 – FIGURE 09.7-1: *Poor R Wave Progression*

Given continued reference by clinicians to “R wave progression” as a component of ECG interpretation — it is essential to appreciate what is meant by the term, “**PRWP**”.

- **PRWP** (*Poor R Wave Progression*) — is said to exist IF the R wave does not progressively increase as one moves across the precordial leads. A number of conditions may be associated with **PRWP**. We list these in *Figure 09.7-1*. The problem is that many of the entities on this list are *clinical “opposites”* (ie, *RVH* vs *LVH*; *normal variant* vs *myocardial infarction*). Clinical utility of a term that *fails* to distinguish between opposites is limited.
- **Clinical Perspective:** The term “PRWP” is clearly suboptimal. That said, *despite* its shortcomings — the designation “**PRWP**” *remains* in active use. Specific indication of *which* precordial leads manifest Q waves (or *QS complexes*) — and noting the area of *transition* is far better. Nevertheless — You’ll want to be aware of the entities commonly associated with PRWP

(**Figure 09.7-1**):

Causes of Poor R Wave Progression (PRWP) :	
—	LVH
—	RVH
—	Pulmonary disease (ie, COPD, chronic asthma)
—	Anterior or anteroseptal infarction
—	Conduction defects (ie, LBBB, LAHB, IVCD)
—	Cardiomyopathy
—	Chest wall deformity
—	Normal variant
—	Lead misplacement (a common cause!)

Figure 09.7-1: Common causes of “**PRWP**” — which is said to exist if the R wave does not become progressively taller as one moves across the precordial leads (See text).

09.8 – **FIGURE 09.8-1: Anterior MI vs Lead Placement Error?**

Technical mishaps and lead placement errors were reviewed in Section 03. Awareness of these technical pitfalls is especially relevant in assessing *precordial* lead R wave progression. For example — Examine the precordial lead sequence shown in **Panel A** of **Figure 09.8-1**.

- Does the precordial lead sequence in **Panel A** make sense? If not — *What might be the cause* of the problem?
- **HINT:** Be sure to look *not only* at R wave progression — *but also* at T wave progression in the precordial leads shown in **Panel A**.

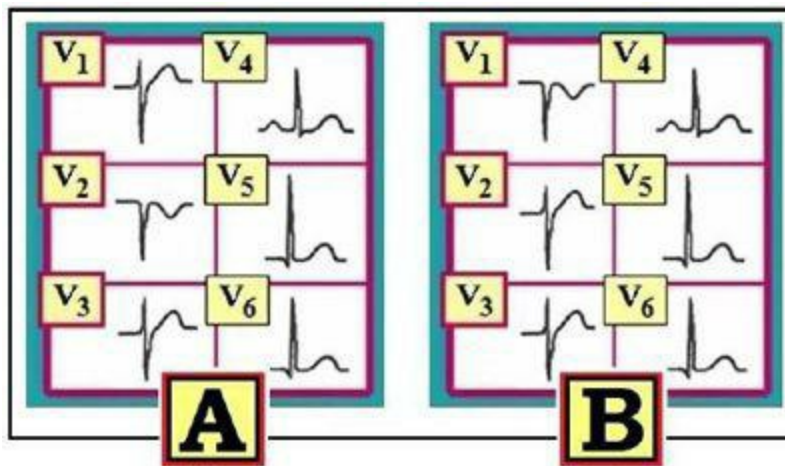


Figure 09.8-1: Precordial lead R wave progression in **Panel A** does not make sense — because there is abrupt **loss and return** of **r** wave *between* V1-to-V3. Note also what happen to T wave morphology as one moves from V1-to-V2-to-V3 in **Panel A** (See text).

Answer to Figure 09.8-1: When R wave progression is “normal” — there is *gradual* increase in R wave amplitude as one moves across successive *precordial* leads. This is not seen in **Panel A** of **Figure 09.8-1**:

- A *relatively* tall initial R wave is seen in lead V1.
- There is **loss of R wave amplitude** between lead V1-to-V2.
- R wave amplitude in Panel A *returns* by lead V3.
- Although possible that abrupt **loss and return** of r wave in **Panel A** could be the result of isolated *anterior* infarction — it is far *more likely* to represent an **error** in **lead placement**. Loss and return of R wave amplitude is usually not nearly *so abrupt* when due to infarction.
- Further support of lead placement error is forthcoming from assessment of changes in T wave morphology. **T wave progression** from lead V1-to-V3 in **Panel A** also does not make sense. Normally — the T wave may be negative, flat (*isoelectric*), or upright in lead V1. The T wave should not go from *positive* in lead V1 — to *negative* in lead V2 — to *positive* once more in lead V3 as it does in Panel A.
- **Putting It All Together:** The precordial lead sequence in **Panel A** would make much more sense IF lead V2 was really lead V1 — and lead V1 was really lead V2. This **technical lead placement error** is in fact the cause of the unusual (*and unrealistic*) R wave and T wave progression seen in Panel A. **Correct lead placement** is shown in **Panel B**. R wave and T wave progression is now normal — with transition *between* lead V3-to-V4.
- **PEARL:** Another reason for *routinely* including assessment of *transition*, *R wave progression* and precordial lead appearance in our **Systematic Approach** (*the “R” in Q-R-S-T Changes*) — is to *avoid* overlooking technical lead placement errors like the one in Figure 09.8-1.

09.9 – FIGURE 09.9-1: What is the Cause of PRWP?

As we have emphasized — the designation **PRWP** (*Poor R Wave Progression*) lacks specificity. That is — *many* conditions may account for relatively *low* anterior r wave amplitude (**Figure 09.7-1**). The purpose of Section 09.9 and Section 09.10 is to illustrate how integration of the **overall ECG picture** facilitates determining *which* of the entities listed in Figure 09.7-1 is likely to be operative.

- The *schematic* precordial lead sequences in *both* **Panel A** and **Panel B** of **Figure 09.9-1** qualify as manifesting “PRWP”. Contemplate *WHY* we label Panel A as suggestive of COPD — and Panel B as suggestive of lead placement error.

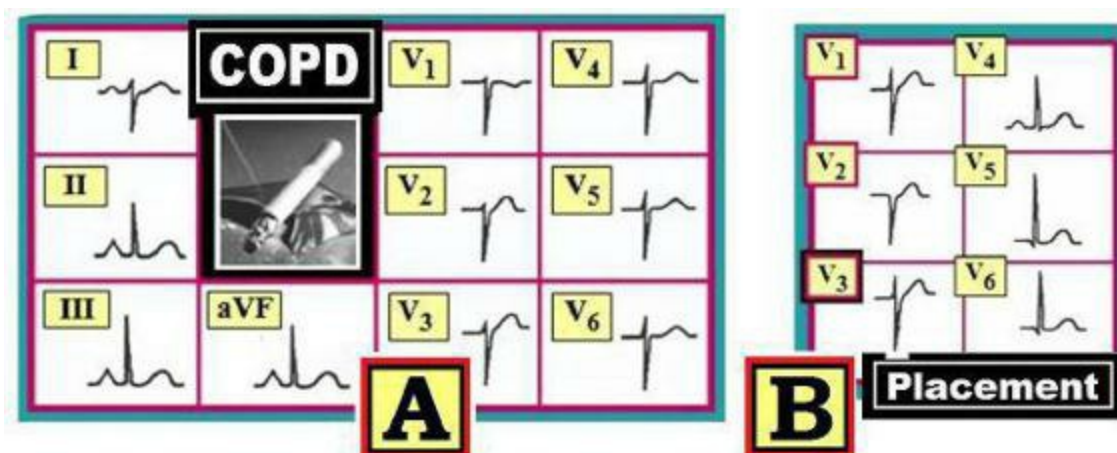


Figure 09.9-1: PRWP due to COPD vs placement error (*See text*).

Answer to Figure 09.9-1: PRWP is clearly present in the *schematic* 12-lead ECG shown in **Panel A**. No more than a *tiny* initial r wave is seen in leads V1,V2 — and R wave amplitude *remains* small through to lead V6. Transition *never* occurs (ie, *the R wave never becomes taller than the S wave is deep*).

- The reason the *schematic* ECG in **Panel A** is *strongly* suggestive of **COPD** — is that *several* additional ECG signs of pulmonary disease are present. These include: **i)** RAD (*Right Axis Deviation*); **ii)** RAA (*Right Atrial Abnormality*); and **iii)** persistent *precordial* S waves that are still deep in V5,V6 (See Section 08.30 for Review of ECG findings with pulmonary disease).
- NOTE: Despite “PRWP” for the *precordial* lead sequence in **Panel A** — **anterior infarction** is *far less likely* because: **i)** an r wave (*albeit a small r wave*) is present in all precordial leads; and **ii)** the *overall* ECG picture in **Panel A** *strongly* suggests **COPD**.
- In contrast to Panel A — the 6 *precordial* leads in **Panel B** strongly suggest **lead placement error**. Abrupt *loss and return* of r wave from V1-to-V3 is *not* a logical sequence for *normal* R wave progression — especially given rapid development of a *predominant* R wave by lead V4. Confirmation of technical error should be easy to establish by verifying chest lead placement and then *repeating* the ECG.

09.10 – FIGURE 09.10-1: QS in V1,V2 vs Anterior MI?

Two final examples of PRWP are shown in Panels B and C of Figure 09.10-1. For comparison — we begin with an example of **normal R wave progression** in **Panel A**.

- Why do we label **Panel B** as suggestive of **anterior MI**?
- Is the precordial lead sequence in **Panel C** also indicative of *septal* and/or *anterior* infarction? If so — *How likely* is it that *prior* infarction has occurred?

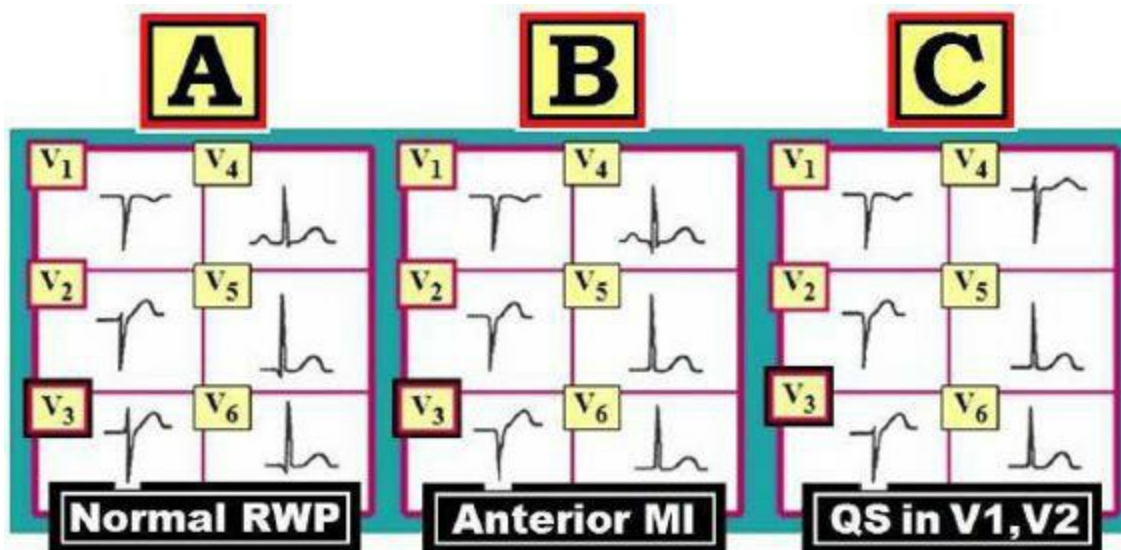


Figure 09.10-1: Normal RWP (*R Wave Progression*) is seen in **Panel A**. This is in comparison with precordial lead sequences suggestive of *anterior* MI (**Panel B**) vs an *indeterminate* pattern showing a QS complex in leads V1,V2 but *not* in V3 (**Panel C**).

Answer to Figure 09.10-1: With *normal* R wave progression (**Panel A**) — there is *progressive* increase in R wave amplitude across *successive* precordial leads until transition occurs. In **Panel A** — the R wave “tops out” in lead V5 and transition occurs *between* V3-to-V4.

- Normally — the QRS complex in **lead V1** is predominantly *negative*, because ventricular depolarization is traveling *away* from this *right-sided* lead. There may (*or may not*) normally be a small upright *initial* r wave in lead V1. Thus, the fact that a QS (*entirely negative*) complex is seen in lead V1 of **Panel A** is completely consistent with *normal* ventricular depolarization.
- Although tiny — a *small* r wave (*positive initial deflection*) is seen by **lead V2** of **Panel A**.
- In contrast, in **Panel C** — a **QS pattern** is seen in *both* **lead V1 and V2**. This may or may not be a “normal” finding. Instead, it could indicate: **i)** a *technical* lead placement error; or **ii)** *septal* infarction. Since there may not be any reliable way to distinguish between normal variant vs lead placement error vs prior *septal* infarction — We simply acknowledge, “**QS in V1,V2; Suggest clinical correlation**” in our interpretation.
- Statistically — the odds are far greater (*approximately 4-to-1*) that *either* normal variant or lead placement error *rather than* septal infarction are the reason for a **QS complex** in **V1,V2** but *not* V3 (*as seen in Panel C*).
- It is only when *no* r wave *at all* is seen in V1,V2 and V3 that prior anterior (*or anteroseptal*) infarction becomes more likely (**Panel B**).
- Additional support that the precordial lead sequence in **Panel B** indicates prior *anteroseptal* infarction — is the finding of a *small-but-definite* q wave in lead V4 but *not* in leads V5,V6. If the initial *negative* deflection in lead V4 was a septal q wave — We would also expect to see *similar* small septal q waves in leads V5,V6.
- **NOTE-1:** The above comments regarding *clinical* implications of a QS complex in leads V1,V2,V3 hold true only when the QRS complex is narrow. QRS widening due to **LBBB** commonly produces QS complexes in anterior leads that are simply the result of the conduction defection and are not indicative of *anterior* infarction (*Section 05.8*).
- **NOTE-2:** There are 2 *other* potential reasons for PRWP with a *narrow* QRS complex that are not the result of prior infarction. These are: **i)** **LVH**; and **ii)** **LAHB**. Usually at least *some* r wave will be seen by lead V3 with these other conditions — though not always. **Bottom Line:** It is sometimes extremely difficult to know with certainty the cause of PRWP when R waves in leads V1,V2,V3 are either very small or absent. *Clinical correlation is essential*.

Beyond-the-Core: It is insightful to appreciate the *physiologic* reason why R wave progression is often “poor” in the presence of LVH *and/or* LAHB.

- **LVH** (*Left Ventricular Hypertrophy*) — produces an increase in leftward and posterior forces. When marked — these leftward and posterior forces may totally *predominate* over initial rightward and anterior forces seen in leads V1,V2,V3. The result is reduction in r wave amplitude in anterior leads. At times — there may even be elimination of any r wave at all in V1,V2,V3.
- **LAHB** (*Left Anterior HemiBlock*) — The bundle of conduction fibers in the LAH (*Left Anterior Hemifascicle*) lies slightly in front of (*anterior to*) fibers in the LPH (*Left Posterior Hemifascicle*). As a result — block in the LAH (*as occurs with LAHB*) results in an initial

posterior direction for electrical activity that is first conducted over the intact LPH. The result may once again be reduction in r wave amplitude in the *anterior* leads.

09.11 – FIGURE 09.11-1: *PRWP from LVH vs Anterior MI?*

Examine the 2 precordial lead sequences shown in Figure 09.11-1. PRWP with *delayed* transition (*between V4-to-V5*) is seen in both **Panel A** and **Panel B**.

- What is the *difference* between these 2 precordial lead sequences?
- Is *anterior* infarction likely in *either* case?
- Does LVH explain all ECG findings?
- Beyond-the-Core: Comment on the ST elevation seen in leads V2,V3 (*short red horizontal lines in leads V2,V3 in Panel B*).

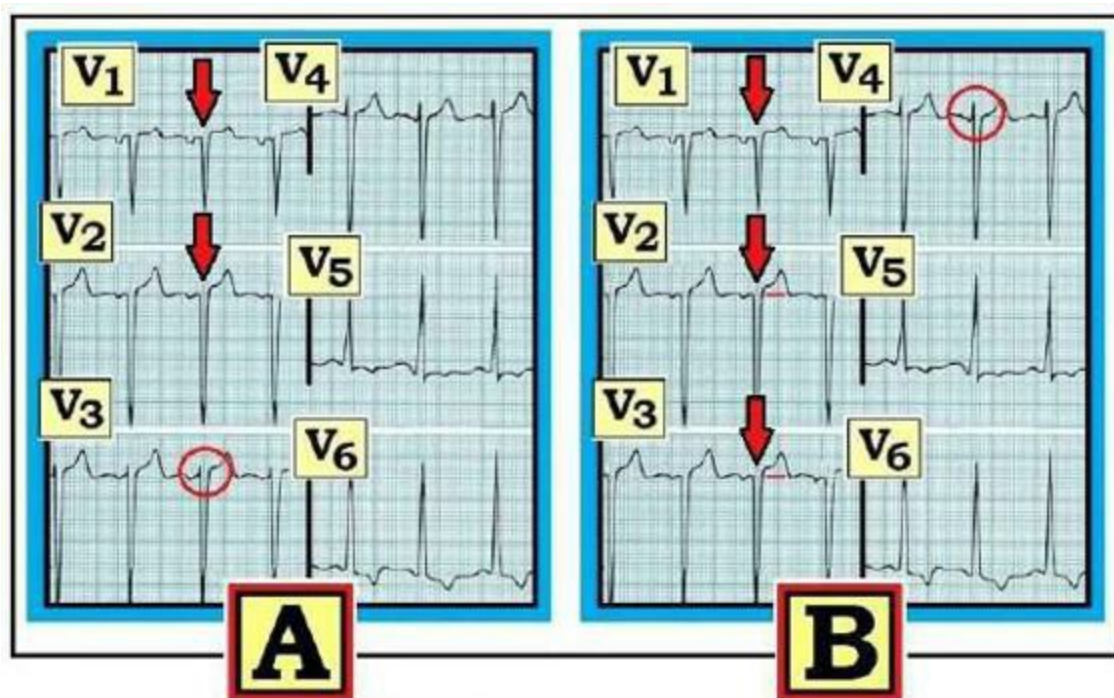


Figure 09.11-1: LVH with PRWP. Red arrows and circles highlight the difference in R wave progression between **Panel A** and **Panel B**. Is there evidence of *anterior* infarction in one or both cases? Does LVH explain *all* ECG findings? (*See text*).

Answer to Figure 09.11-1: QRS amplitude is markedly increased in *both* Panel A and Panel B — easily satisfying criteria for **LVH** (*deepest S in V1,V2 + tallest R in V5,V6 >35mm*). In addition — ST-T wave changes of LV “strain” are seen in lead V6.

- Both precordial lead sequences in Figure 09.11-1 manifest a **QS complex** in leads **V1,V2** (*red arrows*). The difference — is that a QS complex is also present in lead **V3** of **Panel B** (*red arrow*) — whereas a small initial *positive* deflection (*r wave*) is seen in lead V3 of **Panel A** (*red circle*).
- As emphasized in Section 09.10 — the ECG picture in **Panel A** (*QS in V1,V2 but not in lead*

V3) is far more likely not to be due *anterior* infarction. We would therefore write the following as our interpretation: “*LVH and strain; QS in V1,V2 of uncertain significance; Suggest clinical correlation*”.

- In contrast — the ECG picture in **Panel B** (*QS in V1,V2 and in lead V3*) makes it more likely that *anterior* infarction has occurred. That said — QRS amplitude is so greatly increased, that loss of r waves in V1,V2,V3 could be the result of marked LVH. We simply can not know for sure on the basis of this single ECG tracing. We would therefore write the following as our interpretation: “*LVH and strain; QS in V1,V2 and V3; possible prior anterior infarction; Strongly suggest clinical correlation*”.

Final Point: A *subtle-but-real* additional finding in Figure 09.11-1 is the presence of *at least* 2mm of **ST elevation** in leads V2,V3 in *both* Panel A and Panel B (*short red horizontal lines in **Panel B***).

- This ST elevation should be noted. That said — We strongly suspect this ST elevation is not indicative of acute injury because: **i)** Shape of the ST elevation is concave up = “*smiley*” vs ST coving that is far more predictive of acute injury; and ii) the ECG picture in the anterior leads is the reciprocal (*mirror-image*) of what is seen in lead V6. This *mirror-image* picture of concave-up ST elevation in one or more anterior leads is often seen simply as a reflection of LVH in patients without acute infarction.

BOTTOM Line: R wave progression is a *descriptive* finding. There are *many* potential causes of PRWP (*which we listed in Figure 09.7-1*). Some of these causes are benign. Some are not. *Clinical correlation* in context with the *overall* ECG picture is needed to determine likely clinical implications when PRWP is noted.

09.12 – FIGURE 09.12-1: Normal Q Waves; Normal T Inversion

Among the most challenging tasks for the ECG interpreter — is deciding what is “normal” vs “abnormal”. Our purpose in devising **Figure 09.12-1** — is to facilitate remembering when the finding of even a *large* Q wave or *deep* T wave inversion may be normal.

- **5** of the **leads** on a *standard* 12-lead ECG (= *leads III-aVR-aVL-aVF-V1*) — may *normally* display even ***moderate-to-large Q waves and/or T wave inversion*** in *healthy* adults who do not have heart disease.
- Thinking of a “***reverse Z***” (*à la Zorro*) — may help to recall which leads these are (Figure 09.12-1).

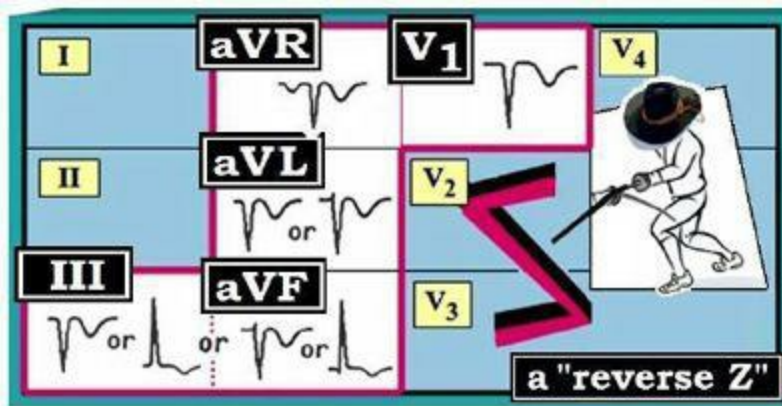


Figure 09.12-1: “Reverse Z” memory aid — for recalling the 5 leads that may occasionally display even *large Q waves and/or T inversion* as an *isolated* finding in otherwise *healthy* adults who do *not* have heart disease (See text).

KEY Points: When are Q Waves/T Inversion a Normal Finding?

Traditionally — the finding of a deep and wide Q wave is thought to be a marker of myocardial infarction. *Other* pathologic reasons for Q waves exist — including cardiomyopathy (*scarring/fibrosis*); conduction defects and WPW. The purpose of this Section is to highlight instances when Q waves (*and associated T wave inversion*) may be a *normal* finding.

- **Normal septal q waves** — are common. As reviewed in discussion of bundle branch block (Section 05.7) — Septal q waves are ***small and narrow***. They arise because the first part of the ventricles to normally depolarize is the *left* side of the septum. As a result — ***left-sided leads*** see the initial depolarization vector as moving *away* from the left as the septum depolarizes from left-to-right. This accounts for the **normal small q wave** that may commonly be seen in one or more of the ***lateral leads*** (= leads I-aVL-V4-V5-V6) in *asymptomatic* individuals *without* heart disease. The reason we do *not* include normal *septal* q waves in Figure 09.12-1 is because their small and narrow dimensions makes it obvious that such q waves are *unlikely* to be pathologic.
- **Lead aVR** — is normally all negative (*negative P wave, QRS and T wave* — as seen in Figure 09.12-1). Global negativity for lead aVR is logical given the remote *right-sided* location of this unipolar lead (*the depolarization vector continually moves away from right-sided lead aVR under normal circumstances*).
- **Lead V1** in adults — typically shows a QS or rS complex *and* T wave inversion (Figure 09.12-1). A **QS complex** may normally *still* be seen in **lead V2** *without* this necessarily meaning there has been prior septal infarction (Section 09.10). However, by **lead V3** — at least *some* r wave should be seen under normal circumstances. Regarding **T wave** appearance in lead V1 — the T should normally be *upright* by lead V2 in adults. Persistent T wave inversion in *anterior* leads should prompt consideration of ischemia. **NOTE:** The situation is *different* in children — for whom *anterior* T wave inversion up to lead V3 (*or even V4*) may reflect a **normal juvenile T wave pattern** (Section 01.4).
- **Leads III, aVL, aVF** (as in Figure 09.12-1) — may all *normally* show T wave inversion *and/or* an *isolated* Q wave that is *not* the result of ischemia or infarction — provided that these findings are not also seen in neighboring leads (See Section 09.13).

09.13 – FIGURE 09.13-1: *Inferior Infarction/Ischemia?*

To illustrate the concept of normal *isolated* Q waves/T inversion — Consider the 6 limb lead sequence shown in **Figure 09.13-1**. Which leads manifest Q waves? Which lead(s) show T wave inversion or ST-T wave flattening?

- Is prior or recent ischemia/infarction likely in **Figure 09.13-1**?

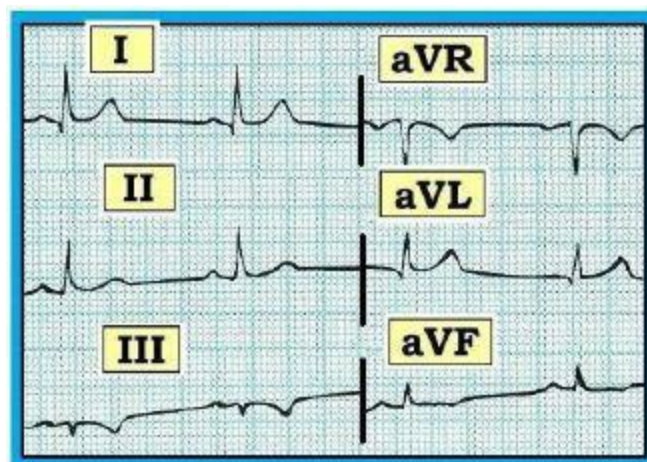


Figure 09.13-1: There is an *isolated* Q wave and T wave inversion in lead III. There are also small q waves in leads I and aVL. How should this be interpreted? (See text).

Answer to Figure 09.13-1: The critical piece of information *missing* from the questions posed about the 6 limb leads shown in **Figure 09.13-1** — is the **history**. Everything seen might *not* necessarily be abnormal — IF the patient was asymptomatic. On the other hand — IF this patient had a history of potentially *worrisome* chest discomfort, the findings in lead III would clearly be of concern. We emphasize the following:

- A **Q wave** is seen in **lead III** of **Figure 09.13-1**. Technically — this is a **QS complex**, since there is *no* R wave. Although this Q wave is not particularly deep (*it is only 2mm*) — given *lack* of any R wave, this Q wave (*QS complex*) portends the *same* clinical implications as would a larger or wider Q wave. That said — Q waves are not seen in the other 2 *inferior* leads (*leads II, aVF*).
- **Figure 09.12-1** tells us — that the finding of an *isolated* Q wave in lead III is *not* necessarily abnormal (*and is not necessarily indicative of prior infarction*).
- There is fairly **deep symmetric T wave inversion** in **lead III** (*especially in view of the small amplitude for the QRS complex in this lead*). IF this patient had *new-onset* chest discomfort — We would clearly be concerned about acute ischemia. That said — there is no more than *nonspecific* ST-T wave flattening in lead aVF and *no* real ST-T wave abnormality in the *other* inferior lead (= *lead II*). **Figure 09.12-1** tells us — that the finding of an *isolated* Q wave *and/or* T wave inversion in lead III (*as well as the nonspecific ST-T wave flattening seen in lead aVF*) is not necessarily ischemic IF there is no ST-T wave abnormality in lead II.
- **NOTE:** The T wave vector often follows fairly close behind the QRS vector. As a result — *isolated* T wave inversion in leads III, aVL or aVF is *less likely* to be ischemic IF the QRS complex is predominantly *negative* in the lead that manifests T wave inversion. This is the case

for lead III in [Figure 09.13-1](#).

- **Bottom Line:** We would note the following on our interpretation: “*Q wave with T inversion in lead III; nonspecific ST-T wave flattening in aVF; Suggest clinical correlation*”. All bets would be off IF this patient had *new-onset* chest discomfort — since absence of abnormality in lead II does *not* completely exclude the possibility of acute ischemia/infarction. But IF the patient was asymptomatic (*especially if a prior tracing was available and showed similar findings*) — We would strongly suspect that the ECG appearance in lead III was not indicative of ischemia/infarction.
- **“Take-Home” Point:** Most of the time when Q waves/T inversion reflects ischemia or infarction — *neighboring* leads (*in this case lead II as well as leads III,aVF*) will show *similar* changes.

What about the Q Waves in Leads I and aVL?

Small narrow q waves are seen in leads I and aVL in [Figure 09.13-1](#). This is a *descriptive* finding that we add to our interpretation at the time we assess for Q-R-S-T changes.

- These **q waves** in **leads I and aVL** of [Fig. 09.13-1](#) are almost certainly **normal septal q waves** given their *small* size and occurrence in association with *no* ST-T wave abnormalities in these high lateral leads (*Section 09.12*).

09.14 – ST Elevation: *Shape/What is the Baseline?*



09.15 – ST Elevation or Depression: *What is the Baseline?*

Determining ST-T wave changes is essential to ECG interpretation. We favor use of the **PR segment baseline** as the reference point for judging ST segment deviations (*elevation or depression*). The **PR segment** is the *connecting line* that extends from the *end* of the P wave — *until* the *beginning* of the QRS complex (**Panel A** in Figure 09.15-1). With this reference point in mind — We define the following:

- **ST Elevation** (**Panel B**) — IF the ST segment arises from *above* the PR segment baseline.
- **ST Depression** (**Panel C**) — IF the ST segment arises from *below* the PR segment baseline.

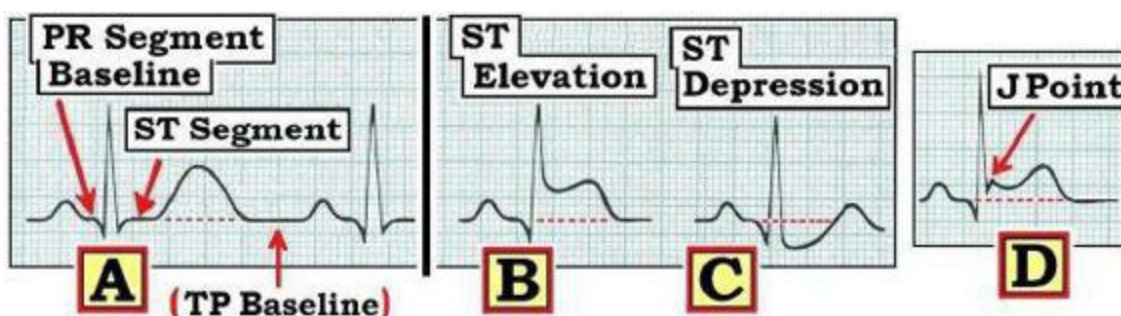


Figure 09.15-1: Use of the PR segment baseline (**Panel A**) — to determine if there is ST elevation (**B**) or ST depression (**C**). Alternatively — *either* the T-P segment or some *combination* of PR and TP segments may be used as the baseline (*See text*). **Panel D** — shows the J-point *notching* that is characteristic of *early repolarization* (*Section 09.20*).

Clinical Notes: Given the importance of the ST segment baseline in determining ST-T wave deviations (*elevation or depression*) — it is important to appreciate the following caveat:

- The **PR segment baseline** — will be more difficult to see when the heart rate is faster. This is because the PR interval *shortens* with **tachycardia**. This makes it more challenging to determine a true “baseline” for the PR segment when the rate is fast. **Other situations** in which assessing the PR segment may be problematic include: **i)** when there is artifact; **ii)** if there is baseline wander; and **iii)** when there is PR depression (*as may occur with acute pericarditis; atrial infarction; and in some normal variants*). Knowing what to use as the baseline for ST segment deviations is sometimes no easy task.

An Alternative Baseline: — Some clinicians favor using the **TP segment** as the baseline for judging ST segment deviations (**Panel A** in Fig. 09.15-1). We feel this is appropriate and a matter of personal

preference.

- **KEY Point:** Far *more* important than whether one selects the TP vs PR segment as the baseline — is to **overview** the *entire tracing* to assess for baseline wander that may affect *whatever* baseline is selected.
- When conditions are ideal (ie, *no baseline wander; no PR depression; all complexes in a lead look identical*) — it is *easy* to pick out the baseline (*and it probably doesn't matter whether you chose the TP or PR as your reference point*).
- **Clinical Reality:** Much of the time — the *optimal* situation of no baseline wander and perfect consistency from *one-beat-to-the-next* is simply not attainable. In such cases — We suggest surveying all 12 leads with goal to *perceive* a “Gestalt” (ie, *to get the best possible “feel” as to what seems to be the isoelectric baseline for the case at hand*). We'll reinforce this concept through numerous clinical examples over remaining Sections in this ePub.

09.16 – J-Point ST Elevation: Recognizing the J-Point

The **J Point** “joins”. It *joins* the *two “S’s”* (ie, *the end of the QRS* — with the **beginning** of the **ST** segment — as shown in **Panel D** of **Figure 09.15-1**). A distinct J point is not always seen (*since the QRS and the ST segment often imperceptibly blend into one another*).

- When it is seen — the **J point** is often *slightly elevated* (*with respect to the PR segment baseline*). This is often a **normal finding** — as will be discussed in detail in the sections on Early Repolarization (*Sections 09.20-through-09.23*).

NOTE: The J point may be **notched** — especially when seen in association with *benign* repolarization variants (**Panel A** vs **Panel B** in **Figure 09.16-1**).

- In contrast — it is *uncommon* for *acute MI* to show J-point notching (*although you may occasionally see notching with acute pericarditis*).

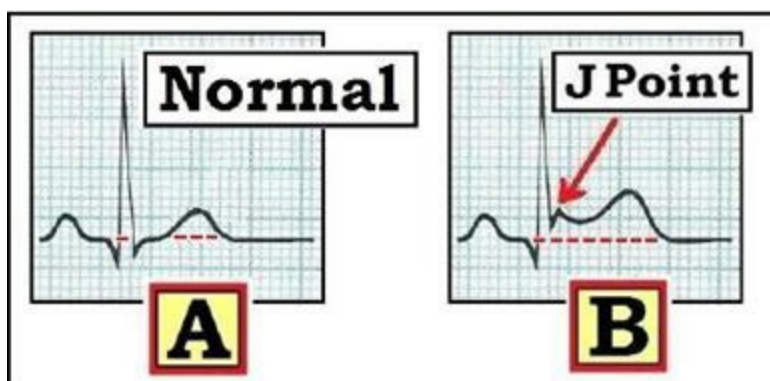


Figure 09.16-1: Comparison of a *normal* ST-T wave which shows *no* ST segment elevation (**Panel A**) — vs the typical J-point appearance (*red arrow*) of patients with normal repolarization variants (**Panel B**). As will be discussed under *Early Repolarization* (*Sections 09.20, 09.21*) — normal

variants often manifest J-point notching and slight ST elevation. Note imperceptible *blending* of the normal ST segment and T wave in Panel A — as distinguished from the **J-point notch** in Panel B that marks the end of the QRS complex and beginning of the ST segment (*See text*).

09.17 – SHAPE of ST Elevation: *More Important than Amount!*

A **KEY** principle to appreciate — is that the **shape** of ST elevation is more important than the *amount* of elevation (**Figure 09.17-1**). For example — *acute* MI may occasionally occur with *no more* than *minimal* ST elevation. There are *other* times — when ST elevation may be much more marked, yet not be due to an acute cardiac event. **Bottom Line:** Appreciation of ST segment **shape** — in context with the clinical situation is essential for accurate interpretation.

- ST elevation with an *upward* concavity (ie, "**smiley**" configuration) is usually benign — especially when seen in an otherwise healthy, *asymptomatic* individual (*and especially when seen with notching of the J point in one or more leads*). This ECG picture represents the benign *normal* variant pattern known as **early repolarization** (**Figure 09.17-1**).
- In contrast — ST segment elevation with **coving** or a *downward* convexity (= "**frowny**" configuration) — is much more likely to be due to acute injury (*from ischemia/infarction*).

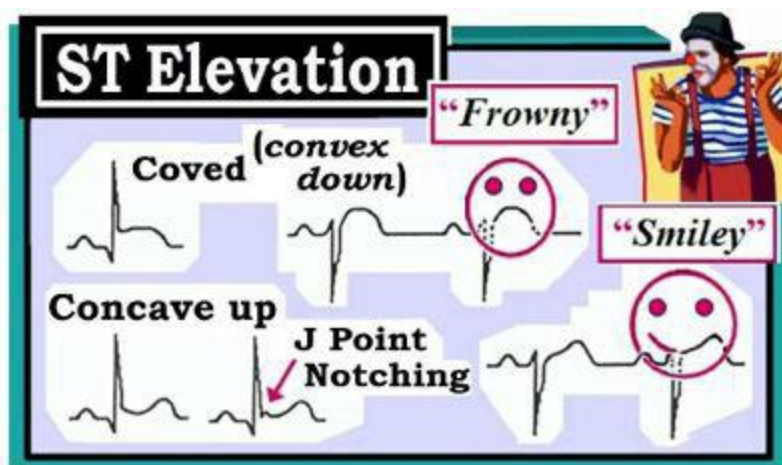


Figure 09.17-1: Shape of ST elevation is *more* important than amount. Upward concavity ("**smiley**"-shape) ST elevation is more likely to reflect the *benign* normal variant of **early repolarization** (*especially if seen with J-point notching*). In contrast — **coved** (= *convex down* or "**frowny**"-shape) ST elevation is more likely to indicate ischemia/infarction (*See text*).

09.18 – HISTORY: *Importance of Clinical Correlation*

History is *ever* important in assessing the clinical significance of ST elevation. Although ST elevation with upward concavity (= "**smiley**" configuration) and J point **notching** will most often reflect a normal *repolarization* variant — this is only true IF the patient is *asymptomatic*.

- In contrast — An *identical* "**smiley-shape**" ST pattern obtained from a patient with *new-onset* chest discomfort must be assumed acute until proven otherwise. IF ever in doubt — *Admit the patient* to the hospital! Look for *old* tracings to compare. *Repeat* the ECG within a *short* period of time. *Don't move on* to your next assignment until you are comfortable that an acute ischemic process is *not* evolving.

- **NOTE:** There is an *uncommon* form of repolarization variant that manifests an ST segment coving (“frowny”) morphology. The clue that this otherwise worrisome pattern is likely to be benign lies in the **History:** The patient is usually a young, healthy athlete (*often African-American*) — who is asymptomatic (See Figure 09.24-1 in *Section 09.24*). Having a prior tracing for comparison may be invaluable. *Without* a prior tracing — it might be *impossible* to distinguish this pattern from *acute* MI if the patient presented to the ED with chest discomfort. **Bottom Line:** The *History* is ever important!

09.19 – FIGURE 09.19-1: *Early Repolarization or Acute MI?*

To solidify the concepts covered thus far — We present the 12-lead ECG shown in Figure 09.19-1. There is definite ST segment elevation in a number of leads. How would you interpret this ECG in the context given for each of the 3 following *clinical* settings?

- **Scenario #1:** — IF the patient was an *asymptomatic* young adult?
- **Scenario #2:** — IF the patient was a *young-to-middle-aged* adult with *recent* URI and *pleuritic* chest pain?
- **Scenario #3:** — IF the patient was an *older* adult with severe *new-onset* chest pain?

NOTE: The **Descriptive Analysis** for ECG findings on this 12-lead tracing does *not* change. Only the **Clinical Impression** changes — depending on which of the above 3 clinical settings is operative.

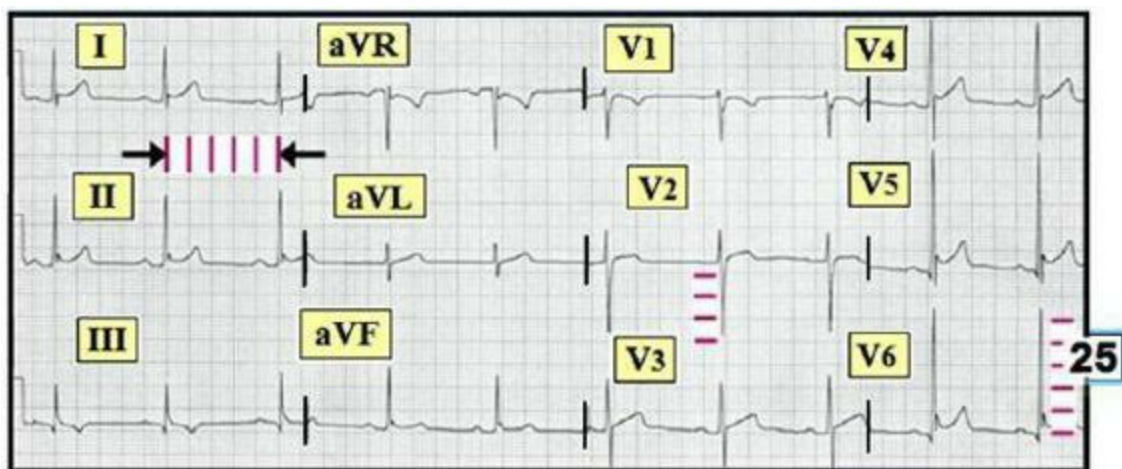


Figure 09.19-1: Sinus rhythm with ST elevation in a number of leads. *What* is the likely cause of ST elevation? (See text).

Descriptive ANALYSIS: The rhythm in Figure 09.19-1 — is sinus at ~60/minute. The PR, QRS, and QT intervals are normal. We estimate the axis at +60 degrees. The sum of the S wave in lead V2 (~3 large boxes = 15 mm) plus the R wave in V5 or V6 (~5 large boxes = 25 mm) clearly *exceeds* 35. This amplitude amount satisfies **voltage criteria** for **LVH** — provided the patient is *at least* 35 years old (*Section 08.1*).

- **QRST Changes** — A **Q wave** and **T wave inversion** is seen in **lead III** — which as *isolated* findings may be normal (*Section 09.12*). There are small *septal* q waves in V5, V6. Transition is

normal (*the R wave becomes taller than the S wave is deep between lead V2-to-V4*).

- The most remarkable finding is **ST elevation** in **multiple leads**. ST segments manifest an *upward concavity* (ie, "**smiley**" configuration). As seen in the *blow-up* of leads V5,V6 (**Figure 09.19-2**) — there is **notching** of the **J-point** (arrow in lead V5). The combination of *upward concavity* ST elevation in association with J-point *notching* — looks very much like the "**smiley**"-shape ST segment appearance of **early repolarization** previously seen in *schematic* Figure 09.17-1.
- Remarkably *absent* from the ECG in Figure 09.19-1 — is *reciprocal* ST depression that would be expected if infarction was acutely evolving.



Figure 09.19-2: Blow-up of leads V5,V6 from Figure 09.19-1. The *upward concavity* ("**smiley**"-shape) ST segment in association with **J-point notching** (arrow) is characteristic of **ERP** (**E**arly **R**epolarization **P**attern). *Diffuseness* of this ST elevation on the 12-lead (Fig. 09.19-1) — *lack* of significant Q waves and *absence* of *reciprocal* ST depression all support ERP as the diagnosis.

Clinical IMPRESSION: The most important point to emphasize about this ECG — is that **our** interpretation of **Figure 09.19-3** will vary tremendously depending on history and the *clinical* setting. **Descriptive Analysis** stays the same. However, what we would do clinically will be very different:

Scenario #1: — IF the patient is an **asymptomatic 25-year old adult**:

- Voltage criteria for LVH would not be satisfied in Figure 09.19-3 (*since the patient is less than 35 years old*).
- We would interpret the ST elevation seen in multiple leads of this tracing as most consistent with **ERP** (**E**arly **R**epolarization **P**attern) — since this young, *asymptomatic* adult manifests benign-appearing *concave up* ST elevation with: **i)** J-point *notching* in at least several of the leads with ST elevation; **ii)** *Lack* of significant Q waves; and **iii)** An *absence* of *reciprocal* ST depression.

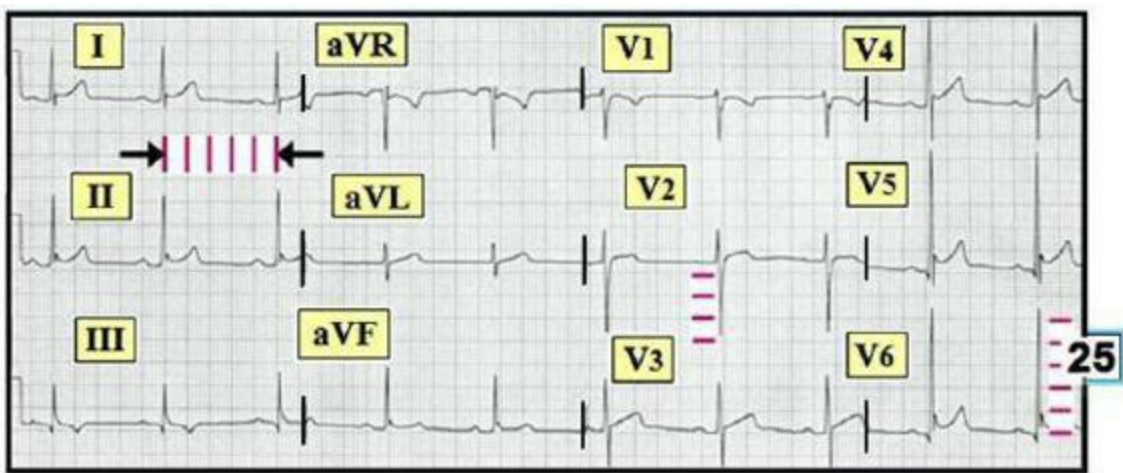


Figure 09.19-3: We reproduce Figure 09.19-1. The likely significance of the diffuse ST elevation seen depends on the clinical setting (*See text*).

Scenario #2: — IF the patient whose ECG is shown in Figure 09.19-3 was a *young-to-middle-aged* adult with history of *recent* URI (*Upper Respiratory Infection*) and **pleuritic** chest pain:

- We would actively *consider* the possibility of **acute pericarditis** — because: **i)** ST elevation is seen in *multiple* leads (*I,II,III,aVF; V2-through-V6*); **ii)** Infarction Q waves are *not* seen; and **iii)** Reciprocal ST depression is absent (*ECG findings of acute pericarditis are discussed in detail in Section 12.2*).

Scenario #3: — IF the patient was *older* with severe **new-onset** chest pain:

- Voltage criteria for LVH now *would* be satisfied.
- We would definitely contemplate the **possibility** of **acute STEMI** (*ST Elevation Myocardial Infarction*) — since there *is* *unmistakable* ST elevation in this patient presenting with *new-onset* chest pain.
- **2-Things-To-Do** — to assist in evaluation would be: **i) Repeat** the ECG in a little while (*to see if there are any evolutionary changes*); and **ii) Look for a prior ECG** — so that you can determine IF the patient has early repolarization as a baseline finding.
- We emphasize that several ECG features in Figure 09.19-3 are **against** this being an **acute STEMI**. These include: **i)** How *diffuse* the ST elevation is; **ii)** *upward* concavity shape with J-point notching; **iii)** the q waves seen are small and narrow; and **iv)** general *lack* of reciprocal ST depression. That said — **the onus of proof is on us** — and, there *is* a Q wave with T inversion in lead III — the ST segment takeoff in lead V3 is *straighter* than is usually seen with early repolarization — and patients with *baseline* ERP *can* develop *superimposed* acute infarction (*so the patient might have had a component of early repolarization prior to developing their acute infarction*).

BOTTOM Line: We *need* to know the *clinical* setting in order to be able to interpret the ECG in Figure 09.19-3. Descriptive analysis will *stay the same regardless* of the history — but clinical interpretation will be vastly *different* depending on what the history is.

Beyond-the-Core: Did you notice the **S1-Q3-T3 pattern** in Figure 09.19-3? It turns out that this ECG was obtained from a healthy, *asymptomatic* young adult and that the ST elevation was entirely due to *early repolarization*.

- As emphasized in Section 08.36 — the *isolated* finding of an **S1-Q3-T3** pattern in an otherwise healthy individual may be normal. An S1-Q3-T3 is only helpful in the diagnosis of *acute PE* (*Pulmonary Embolus*) — IF the history is “*right*” and there are *other* suggestive ECG findings.

09.20 – What is **EARLY REPOLARIZATION**?

Having discussed the elements required for determining whether there is ST elevation — with emphasis on the importance of assessing ST segment *shape* plus the *clinical* history — we now explore further the entity known as **ERP** (*Early Repolarization Pattern*).

- The term **ERP** — relates to a common ECG finding characterized by **J-point abnormalities** and *associated ST-T wave changes*. These features are shown in **Figure 09.20-1**:
- Typically — there is J-point ST elevation with *upward* concavity (**Panel A**).
- There may be frank **notching** — defined as an upward deflection near the end of the R wave downslope (**Panel B**).
- Alternatively — there may be **slurring** (*without notching*) of the terminal portion of R wave downslope as it transitions into the ST segment (**Panel C**). Note that there is no more than minimal ST elevation in association with such slurring in Panel C.

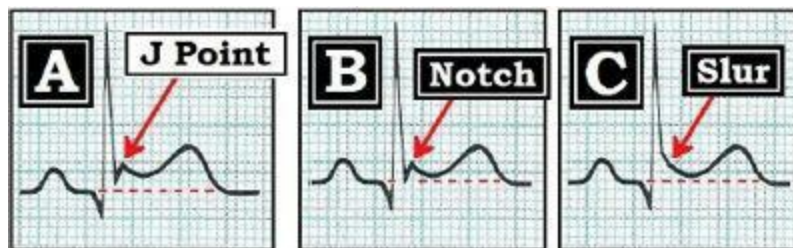


Figure 09.20-1: ECG features of *early repolarization*. There is *concave up* ST elevation (“*smiley*”-*shape*) in at least *several* leads — often with either J-point notching and/or terminal R wave slurring in one or more of the leads that manifest ST elevation (*See text*).

09.21 – *Early Repolarization: Variations in the Definition*

First described in the 1940’s — there are *variations* in **how** even the **experts define “ERP”**. Many imply that the entity is defined by J-point ST elevation of *characteristic* shape. Others include J-point abnormalities (*notching, slurring*) — even when there is no more than *minimal* associated ST segment elevation.

- Typically — there is at least **1-2 mm** of *upward concavity* (“*smiley*”-*shape*) **ST segment elevation** with prominent **upright T waves** in *at least 2 contiguous leads*.
- Some include *anterior* lead ST elevation within their definition of “ERP”. Others maintain that *some degree* (*1-2mm*) of *anterior* ST elevation (*in V2-to-V4*) is so common as to be a **normal**

finding (and not indicative of ERP).

- The presence of 1-2mm *upward* concavity **ST elevation** in *either inferior (II,III,aVF) or lateral (I,aVL; V4,V5,V6) lead areas* is much **less common** than *anterior* ST elevation – and – *clearly* satisfies criteria for “ERP”.
- *More than one lead area may be involved* with ERP. Occasionally there may even be “*global*” ST elevation (*in inferior, lateral, and anterior leads*) — which may understandably be *difficult to distinguish* from *acute* pericarditis.
- **ECG changes** of ERP may be **dynamic**. They may come or go with *change* in body position (*from supine to standing*) — with activity (*they commonly disappear during exercise testing*) — and ERP may change over time (*especially if the patient becomes deconditioned or during illness*). **CAVEAT:** Such ST-T wave changes do not necessarily indicate ischemia or *acute* infarction!
- **Other Patterns** of “**repolarization variants**” exist. These include *anterior* T wave peaking (Figure 09.23-1) — and ST segment coving (Fig 09.24-1).

09.22 – ERP: Is Early Repolarization Benign?

In years past — the *incidental* finding of *upward concavity* ST elevation in an otherwise healthy and asymptomatic *young-to-middle-aged* adult was thought to be benign. As a result — the name of this entity evolved to the term, “**BER**” = **B**enign **E**arly **R**epolarization. While fully agreeing on the overall favorable prognosis for the overwhelming majority of patients with this type of ST elevation — We feel “**ERP**” (**E**arly **R**epolarization **P**attern) is a **far better term** than “BER” because:

- The *incidental* ECG finding of **early repolarization** in *young-to-middle-aged* asymptomatic adults is **not necessarily benign**. Tikkanen et al (*NEJM* 361:2529, 2009) — have shown a 1.3-fold *increased* risk of cardiac death with ≥ 1 mm early repolarization ST elevation (*and up to a 3-fold increased risk IF > 2 mm ST elevation*). **What to do with these results?**

The Facts about ERP are the following:

- Early repolarization is common. It is seen in *at least* 2-5% of the general population. Cardiac arrest is *rare* in these patients. *Most patients* with *incidental* ERP on ECG have a *benign* prognosis.
- IF the patient is otherwise healthy and asymptomatic — We choose not to be concerned by the *incidental* finding of early repolarization on a baseline tracing.
- There is general consensus that the *isolated* finding of *anterior* ST elevation conveys *no* increase in cardiovascular risk. This form of ERP is benign.
- *Final* answers are not yet “in” for *inferior or lateral* ERP. **Consider further evaluation IF:** i) there is a *positive* family history of *sudden* death at *early* age; ii) the patient had *unexplained* syncope/presyncope; and/or iii) there are *other* symptoms, arrhythmias or concerns.

09.23 – FIGURE 09.23-1: *Acute MI or Repolarization Variant?*

Illustration of how best to interpret ECG findings suggestive of ERP is best accomplished by clinical example. Consider the ECG shown in **Figure 09.23-1** — which was obtained from a 50-year old man with *atypical* chest discomfort.

- Is this ECG likely to reflect ERP or acute *anterior* MI?
- How *confident* are you of your answer?
- Are you *comfortable enough* to send this patient home IF he presented to an ED (*Emergency Department*) with this tracing?
- Would you send the patient home IF he presented to the office with this ECG and the *same* history of *atypical* chest discomfort?

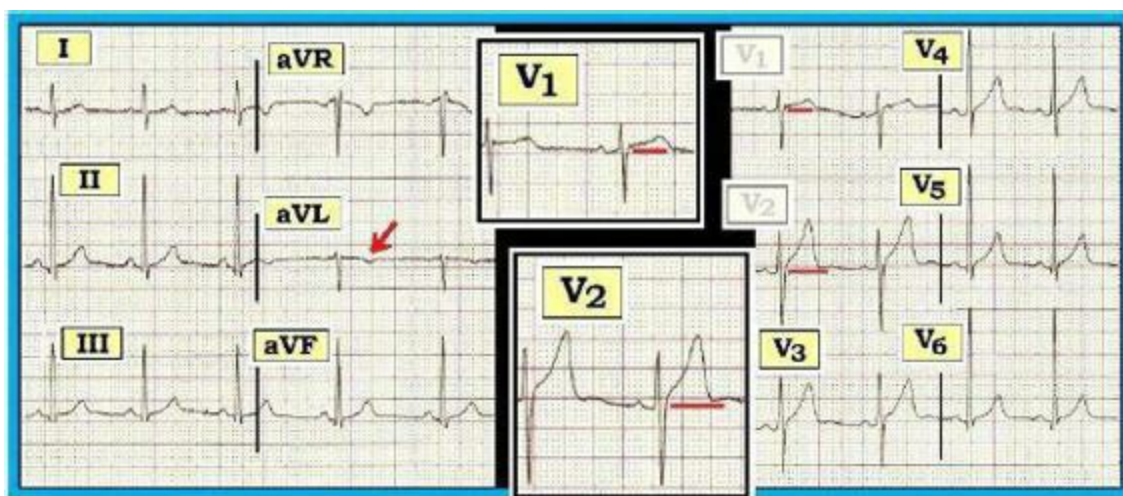


Figure 09.23-1: ECG obtained from a 50-year old man with *atypical* chest discomfort. Is the ST elevation seen in leads V1,V2 likely to represent an *early acute anterior* STEMI? How *confident* are you of your answer? (See text).

Answer to Figure 09.23-1: Descriptive analysis of this ECG reveals the rhythm to be sinus arrhythmia. Intervals and the axis are normal. There is *voltage* for LVH (*deepest S wave in V1,V2 + tallest R wave in V5,V6* ≥ 35).

- **QRST Changes** — There are small q waves in the inferior and lateral precordial leads. R wave progression is normal (ie, *transition occurs between leads V3-to-V4*). **T waves are peaked.** There is some **J-point ST elevation** (*with upward concavity = “smiley” configuration*) in multiple leads — and there is **shallow symmetric T wave inversion** in lead aVL (red arrow).

IMPRESSION — Accurate clinical interpretation of this tracing depends on the history. Without any history — We would not know clinically how to proceed.

- **IF concerned** that the history of “*atypical chest discomfort*” in this 50-year old man sounded like angina (*especially if his symptoms were of new-onset*) — the patient should be *admitted* to the hospital! There is **precordial lead ST elevation**. We would: **i)** Look for a **prior tracing** for comparison (*to see if there was change from his baseline ECG*); and **ii)** **Repeat** the ECG in

short order — to see if there is acute evolution.

- That said — We **strongly suspect ERP** as the cause of ST elevation in Figure 09.23-1 because:
 - i) ST elevation manifests an *upward* concavity = “*smiley*” configuration; and ii) The *lack* of reciprocal ST depression. Although there is shallow T wave inversion in lead aVL — this is probably a normal finding given predominant negativity of the QRS complex in this lead and lack of associated T inversion in other lateral leads (*Section 09.12*).
- ***Beyond-the-Core:*** Several *additional* ECG features in Figure 09.23-1 are present that ***favor ERP*** (*rather than acute infarction*). These include the following: i) that precordial lead QRS amplitude is increased (*increased R wave amplitude in V1-through-V4 is less likely when there is anterior infarction*); ii) that the R wave is *already* fairly tall by V2,V3 (*delayed transition is commonly seen with anterior infarction*); and iii) that the QT is definitely *not* long (*acute ischemia/infarction commonly results in at least some lengthening of the QT interval*). That said — the ***clinical history*** — a ***repeat ECG*** and availability of ***prior ECGs*** for ***comparison*** will ultimately dictate the course.
- ***NOTE:*** *Where* the patient presents for evaluation *does* have a role in determining the likelihood of being admitted to the hospital. Given *identical* clinical features and a *similar* ECG — the likelihood of being admitted to the hospital appears to be *more* ***IF*** the patient is seen in an ED (*Emergency Department*). This is because: i) Emergency care providers are less likely to know the patient and the previous medical history (*and also less likely to have a prior ECG available for comparison*); and ii) There appears to be a *tendency* toward ***self-selection***, whereby patients with more acute cardiac disorders *somehow* know to go to the ED rather than the office. That said — ***the onus remains on us*** to assume the worst scenario (ie, ACS = *Acute Coronary Syndrome*) until we can be comfortable that it is safe to send the patient home. ***BOTTOM Line:*** The clinical history is needed in order to know what to do for a patient presenting with the ECG shown in Figure 09.23-1. While a diagnosis of ERP *without* need for admission would be reasonable ***IF*** the history was *not* of concern — a very *different* conclusion would be reached ***IF*** symptoms sounded acute.

T Wave Peaking: The final point to make from the ECG in Figure 09.23-1 — is that ***repolarization variants*** that occur in otherwise healthy adults may take on a number of different forms. These include: i) Upward concavity (“*smiley*”-shape) ST elevation (*Sections 09.20 and 09.21*); ii) ***T wave peaking*** (Figure 09.23-1); and iii) *Other patterns* (*See Section 09.24*).

- The ECG finding of ***peaked T waves*** should prompt consideration of ***3 entities***. These include: i) Hyperkalemia; ii) Ischemia; and iii) A ***normal repolarization variant***. Morphologic appearance, clinical correlation and comparison with *prior* tracings may be needed to distinguish between these possibilities.
- ***Against*** hyperkalemia in Figure 09.23-1 — is that T waves are *not* pointed; the ascending and descending limbs of the T waves are *not* symmetric; and the base of the T waves is *not* narrow (*Section 11.3*). Patients with hyperkalemia almost always have either a *predisposing* medical illness (*severe renal disease; acidosis; dehydration; multi-organ failure*) or are taking *potassium-retaining* medications.
- ***Posterior ischemia*** — may manifest as *anterior* T wave peaking. That said, ischemia is highly

unlikely — IF T wave peaking is diffuse and the patient is a younger adult who is *not* having chest pain (*as occurs in this case*).

- **T wave peaking** (*as seen in Figure 09.23-1*) — is a *descriptive* finding. We presume it is due to a **benign repolarization variant** only after we are comfortable that it is not due to hyperkalemia or ischemia.

09.24 – FIGURE 09.24-1: *Acute MI or Repolarization Variant?*

Consider the ECG shown in **Figure 09.24-1**. No history is given.

- Is this ECG likely to reflect ACS (*A*cute *C*oronary *S*yndrome) with possible recent infarction?
- Can you think of a clinical scenario whereby the ECG shown in **Figure 09.24-1** might be a *normal* variant?
- Extra Credit: Is there LVH?

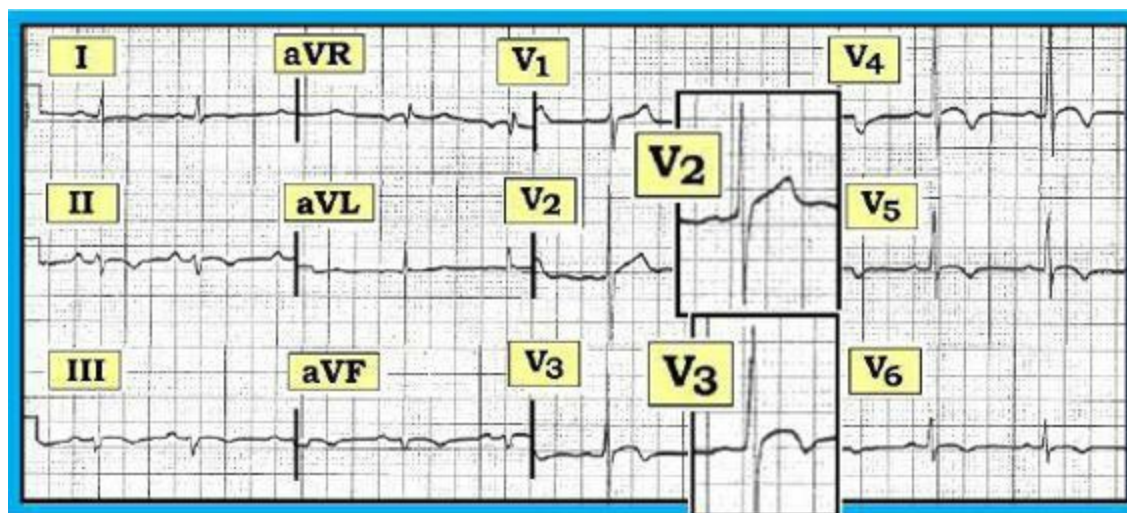


Figure 09.24-1: No clinical history is given. Has there been *recent* infarction (*or ongoing acute ischemia*)? Does this patient have LVH? What information is needed to answer these questions? (*See text*).

Answer to Figure 09.24-1: Descriptive analysis of this ECG reveals the rhythm to be sinus arrhythmia. Intervals are normal. There is **marked LAD** (*L*eft *A*xis *D*eviation) that satisfies criteria for **LAHB** (*L*eft *A*nterior *H*emi*B*lock) — as the *net* QRS deflection in lead II is predominantly negative (*Section 07.13*).

- **Chamber Enlargement** — Did you notice there is **half standardization**? The standardization mark is seen at the very beginning of the tracing — and is only 1 *large* box (5mm) tall (*red rectangle in lead II of Figure 09.24-2*). This means that amplitude of the P wave and QRST complex has been reduced in half in order to fit on the ECG recording paper (*Section 08.5*). Therefore — **S wave** depth in **lead V2** is not 12 mm, but **24 mm** — and **R wave** amplitude in **lead V5** is not 10 mm, but **20 mm**! Therefore — **voltage criteria** for LVH will be satisfied IF the patient is 35 years old.

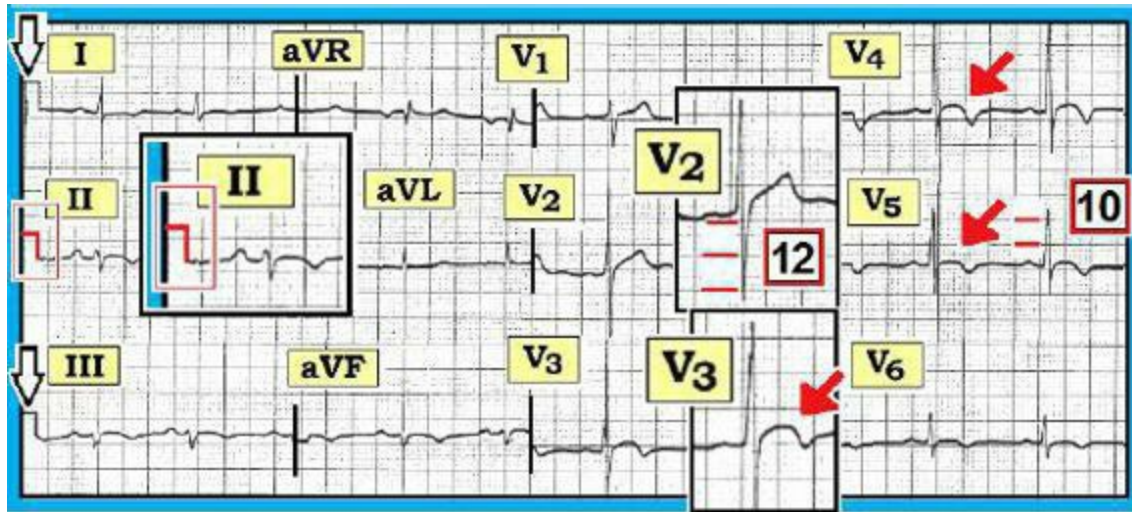


Figure 09.24-2: The ECG shown in [Figure 09.24-1](#) is reproduced — highlighting that there is *half standardization* (red rectangle in lead II; open arrows at the very beginning of leads I and III). As a result — **voltage** for LVH is present IF this patient is 35 years old. Are you concerned about the ST-T waves in the *inferior leads* and especially in leads V3-through-V6 (red arrows)?

Assessment of QRST Changes: No Q waves are present in [Figure 09.24-2](#) — as *small-but-real* initial r waves are seen in each of the inferior leads. Transition occurs early due to a surprisingly tall R wave in lead V2. That said — the most concerning finding on this ECG is **ST coving** with *slight* **ST elevation** and **T wave inversion** in *multiple* leads (red arrows in precordial leads).

- IF this patient was older with a history of *new* chest pain — this ECG would look like an *acute* evolving MI. It turned out that the patient in this case was a young adult athlete with *no* heart disease who had this ECG done as part of a pre-participation sports exam. Thus, the ST-T wave changes seen are most likely to represent an *unusual repolarization variant*. In view of this benign clinical setting — there is no evidence for acute ischemia. **Bottom Line:** Be aware that there are a number of different repolarization variants that may be seen on the ECG of *normal* subjects. **History is everything!**
- ***Beyond-the-Core:*** If we were evaluating this asymptomatic young adult athlete in the office as part of a *pre-participation* exam — We would recommend an **Echo** to *rule out* hypertrophic cardiomyopathy given early transition with prominent septal forces (ie, *disproportionately tall* R waves in leads V2, V3). If physical examination and the Echo were normal — there would be no reason not to allow full participation.

09.25 – ST Segment Depression



As discussed in Section 09.15 — We favor use of the **PR segment baseline** as the reference point for judging ST segment deviations (*elevation or depression*). The **PR segment** is the **connecting line** that extends from the **end** of the P wave — until the **beginning** of the QRS complex (**Panel A** in Figure 09.25-1). Using the PR baseline as our reference point — We define the following:

- **ST Elevation** (**Panel B**) — IF the ST segment arises above the PR baseline.
- **ST Depression** (**Panel C**) — IF the ST segment arises below the PR baseline.

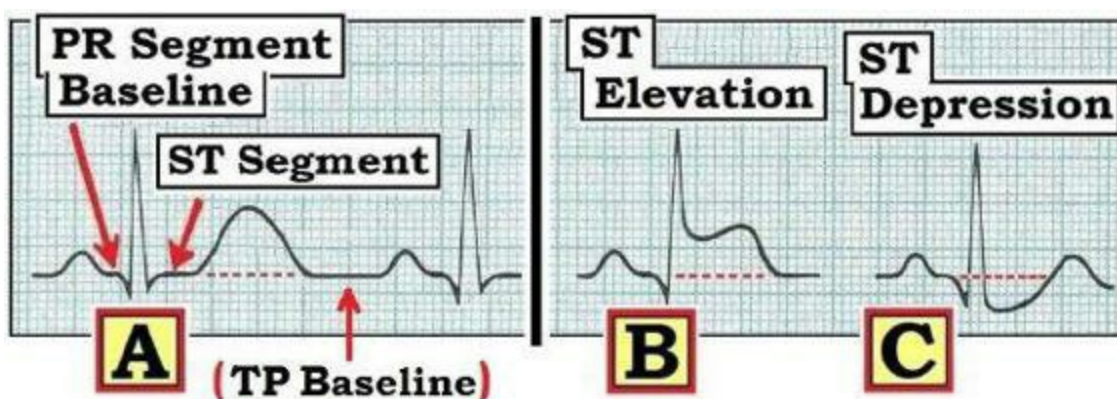


Figure 09.25-1: Use of the PR segment baseline (**Panel A**) — to determine if there is ST elevation (**B**) or ST depression (**C**). Alternatively — either the T-P segment or some *combination* of PR and TP segments may be used as the baseline (*See text*).

Clinical Caveats: The **PR segment baseline** is more difficult to see when the heart rate is faster. This is because the PR interval *shortens* with tachycardia. This makes it more challenging to determine a true “baseline” for the PR segment when the rate is fast.

- *Other* situations in which assessing the PR segment may be problematic include presence of artifact and baseline wander. Knowing what to use as the baseline for ST segment deviations is sometimes no easy task.
- **NOTE:** Instead of the PR segment — some clinicians favor the **TP segment** as the baseline to use for judging ST segment deviations (**Panel A** in Figure 09.25-1). *Either* approach is acceptable — and, when conditions are ideal (ie, *no artifact; no baseline wander; all complexes in a lead look identical*) — determining the baseline is easy (*and it doesn't really matter whether you select the TP, PR, or some combination of the two as your reference point*).

09.26 – LIST #4: Causes of ST Depression

There are **over 50 causes** of ST segment depression on ECG. Many of these causes are *cardiac-*

related — but there are also many *non-cardiac* causes. Among the many ***non-cardiac*** causes are hyperventilation; temperature extremes (*excessive heat or cold exposure*); anxiety or emotional stress; anemia; tachycardia; sleep deprivation; pulmonary disease; electrolyte abnormalities; central nervous system disorders; certain medications; and severe medical illness (*among others*). **BOTTOM Line:** Rather than commit to memory an *exhaustive* list of conditions predisposing to ST segment depression — it suffices to appreciate the variety of entities that may produce changes on ECG. **Remember:** *Many of these conditions are non-cardiac.*

- We *simplify* recall of those clinical entities predisposing to ST depression that we feel are *most* important to remember with our **LIST #4** in **Figure 09.26-1**.
- In general — **causes of T wave abnormalities** (including *T wave flattening or frank T wave inversion*) are *similar* to potential causes of ST segment depression.

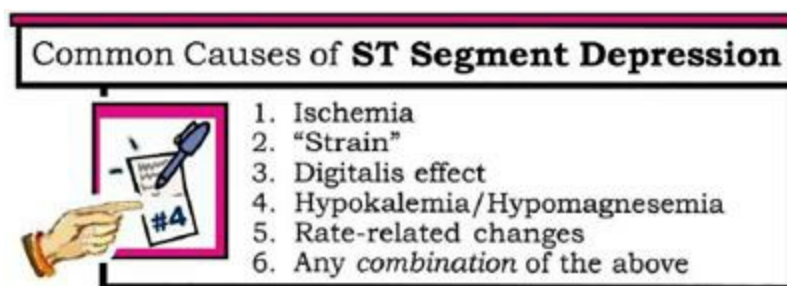


Figure 09.26-1: Common Causes of ST Depression = **LIST #4**. A similar list may produce T wave changes. *More* than a single cause may be operative in a given patient.

09.27 – ST-T Wave Appearance: *A Hint to the Cause*

Although *all* 6 of the **possibilities** noted in **List #4** should be considered *each* time you encounter ST-T wave abnormalities — the *specific* cause(s) operative may be suggested by **appearance** of the ST-T wave on ECG (**Figure 09.27-1**):

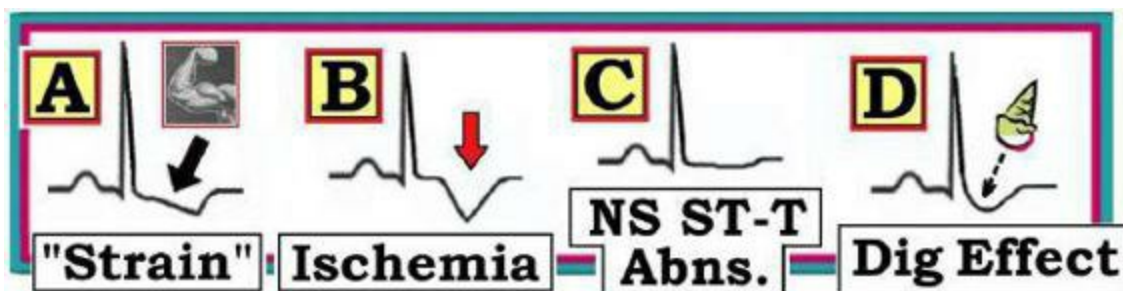


Figure 09.27-1: ST-T wave *appearance* may suggest the cause(s) operative in a given patient (*See text*).

Shape of the ST-T wave — may suggest which of the 6 entities in **List #4** is likely to be operative in a given patient (**Figure 09.27-1**):

- **Ischemia** — is suggested by **symmetric T inversion** (*red arrow in Panel B*) — especially when seen in two or more leads of a given lead group (ie, in leads II, III and aVF — or in both leads I and aVL).
- In contrast — **"Strain"** from LVH (**Panel A**) is suggested by **asymmetric ST depression**

occurring in one (*or more*) of the **lateral leads** (*less often in the inferior leads*). Note the initial *slow* sagging of the ST segment with “strain” (*black arrow*) — with more rapid return to the baseline (*Section 08.9*). “Strain” is more likely to be the cause of ST depression when voltage for LVH is present — although occasionally, there may be ST-T wave changes suggestive of “strain” *without* accompanying voltage (*Section 08.10*).

- **"RV strain"** — is suggested IF the picture in **Panel A** of Fig. 09.27-1 is seen in **right-sided leads** (*II, III, aVF or V1, V2, V3*) in a patient with **RVH** (*Sections 08.28 and 08.29*).
- Use of **Digoxin** may **affect ST-T waves** in one of **3** ways: **i)** there may be **“scooped” ST depression** in multiple leads (*that simulates an inverted ice cream cone, as in Panel D of Figure 09.27-1*); **or ii)** Digoxin “effect” may produce a **“strain”-like pattern** (*identical to Panel A*); **or iii)** Despite use of Digoxin — ST-T waves may be unaffected. It is of interest that the serum Digoxin level does *not* correlate well with ST-T wave appearance in a patient taking this drug. Thus, a patient may manifest classic “scoopies” (*scooped ST depression with a short QT interval in multiple leads*) — yet be on no more than a *low* dose of the drug. In contrast, another patient may have toxic serum levels of Digoxin — yet manifest *no* ST-T wave abnormalities at all. **Bottom Line:** ST-T wave appearance does *not* predict which patients have Digoxin toxicity.
- **Panel C** — is the remaining pattern of ST-T wave change in Figure 09.27-1. We label this pattern as, **“NS ST-T Abns.”** (= ***Non-Specific ST-T wave Abnormalities***) — since such ST flattening *and/or* slight depression does *not* suggest any *specific* cause. Instead — *any* of the *more* than 50 potential causes of ST-T wave abnormalities may be contributing to the appearance of this pattern. **Bottom Line:** The ST-T wave is *not* normal — but the change that we see is **non-specific** in that we *don’t* know the cause.

09.28 – FIGURE 09.28-1: *What is the Cause(s) of ST Depression?*

It is easiest to illustrate the concepts covered in Sections 09.26 and 09.27 by a clinical case. Interpret the ECG shown in **Figure 09.28-1** — obtained from a woman on *multiple* medications. She has chest pain and heart failure. Note the *marked* amount of **ST depression** in **multiple leads** — which is especially well seen in the blow-up inserts from leads V5, V6 (*red arrows*).

- In view of this clinical history — What are likely causes of this marked amount of ST depression?
- **HINT:** Feel free to refer back to **LIST #4** in formulating your answer (Figure 09.26-1).

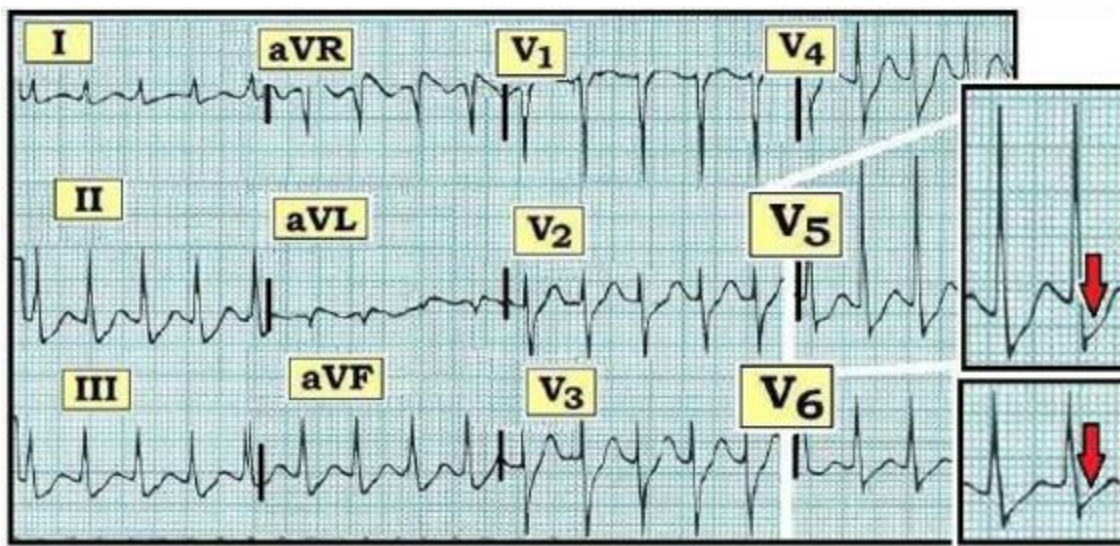


Figure 09.28-1: ECG obtained from a woman with chest pain and heart failure. She is taking *multiple* medications. What are *likely* causes of the *marked* ST depression seen in *multiple* leads? (Feel free to refer back to **List #4** in Figure 09.26-1 when formulating your answer).

Answer to Figure 09.28-1: The rhythm is an almost **regular SVT** (*narrow complex tachycardia*) at ~180/minute. Given the rate and the lack of any definite P waves — we presume **PSVT** for the rhythm diagnosis (**List #2** — in Section 02.52). The axis is normal; there is voltage for LVH.

- The most remarkable finding on this 12-lead ECG is marked and **diffuse ST depression**. ST segments are depressed *at least* 3mm below the J-point baseline in *multiple* leads (*seen especially well in the blow-up inserts of leads V5, V6 — red arrows in Fig. 09.28-1*).
- Recognition of the ECG finding of **ST depression** should prompt consideration of the clinical entities noted in **List #4** (*in Figure 09.26-1*).
- The **clinical History** allows us to hone in on the likely causes of ST depression. In this case — *any (probably several)* of the entities in **List #4** may be operative, since this patient has chest pain (*possible ischemia*) — she has heart failure and LVH (*LV “strain” is probably present*) — she is on lots of drugs (*possibly digoxin; probably diuretics predisposing to low K⁺/Mg⁺⁺*) — and she is tachycardic (*SVT rhythm at ~180/minute*).

“Take-Home” Point: The ECG in Figure 09.28-1 provides an excellent example of how best to utilize **List #4**. We interpret the 12-lead tracing in the usual systematic fashion. Recognition of **diffuse ST depression** should set off the “light bulb” to recall the entities on List #4. Clinical correlation with the history given allows us to predict a likely contribution from each of the entities on the list.

09.29 – Recognizing *Subtle* ST Changes: **ST Segment Straightening**

Consensus among expert electrocardiographers is lacking regarding the definition of a “normal” ECG. Much of this relates to semantics — since *minor* ST-T wave abnormalities generally provide no more than a *nonspecific* suggestion to potential etiologies. That said — We feel it is important to **hone in** on recognizing even *minimal* abnormalities, if for no other reason than to let those reading our interpretation be aware that *we saw the abnormality* in question but did *not* think it clinically important for the case at hand.

- The above said — there are times when even *minimal* ST-T wave changes may have clinical relevance. In addition — *routine* attention to **recognizing subtle ST-T wave changes** will go a *long* way toward improving one's ECG interpretation ability.
- For example — *What is the difference* between the ST segment shown in **Panel A** vs **Panel B** in Figure 09.29-1? Is the admittedly *subtle* difference in ST-T wave appearance between these two complexes likely to be of clinical significance? If so — *How*?

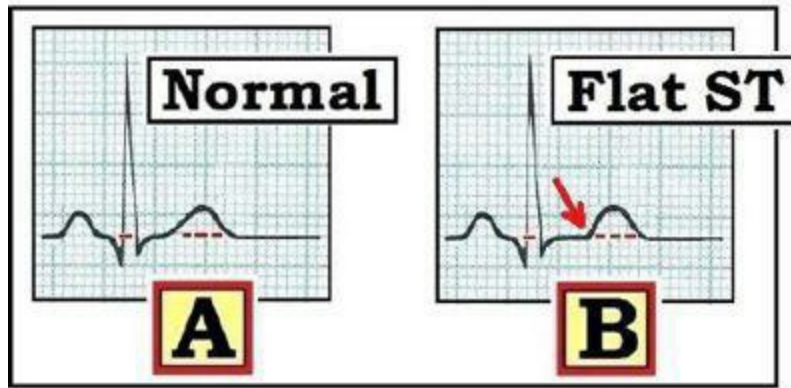


Figure 09.29-1: Compare the ST segment in **Panel A** with **Panel B**. *What is the difference?* Is this likely to be clinically significant? (See text).

Answer to Figure 09.29-1: The ST-T wave in **Panel A** is *normal*. Note the *smooth* contour at the point of transition between the end of the S wave and the beginning of the ST segment. Note an equally *smooth* contour at the end of the ST segment and the point where the ascending limb of the T wave begins.

- In contrast — Note the **sharp angle** in **Panel B** at the point where the straight (*flat*) ST segment ends and the ascending limb of the T wave begins (*red arrow*). While admittedly “splitting hairs” — the ST-T wave in **Panel B** is *not* normal. Instead — there is **nonspecific ST segment straightening** (ie, *loss of that smooth transition between end of the ST segment and the beginning of the T wave ascending limb*).
- We emphasize that “*nonspecific ST segment straightening*” — is a **descriptive finding**. It is *nonspecific*. It may mean nothing — especially if only seen in a single lead. In any case — it is *not* an acute change. On the other hand — ST segment *straightening* as occurs in **Panel B** may at times be a *nonspecific* indicator of underlying coronary disease — especially when this finding is seen in more than one lead. *Clinical correlation is everything*.

09.30 – FIGURE 09.30-1: *Are the ST Segments Normal?*

Interpret the ECG shown in Figure 09.30-1 — obtained from an adult with a recent history of *atypical* chest discomfort.

- Would you classify the ECG shown in **Figure 09.30-1** as a “normal” tracing?
- If not — *Why not?*

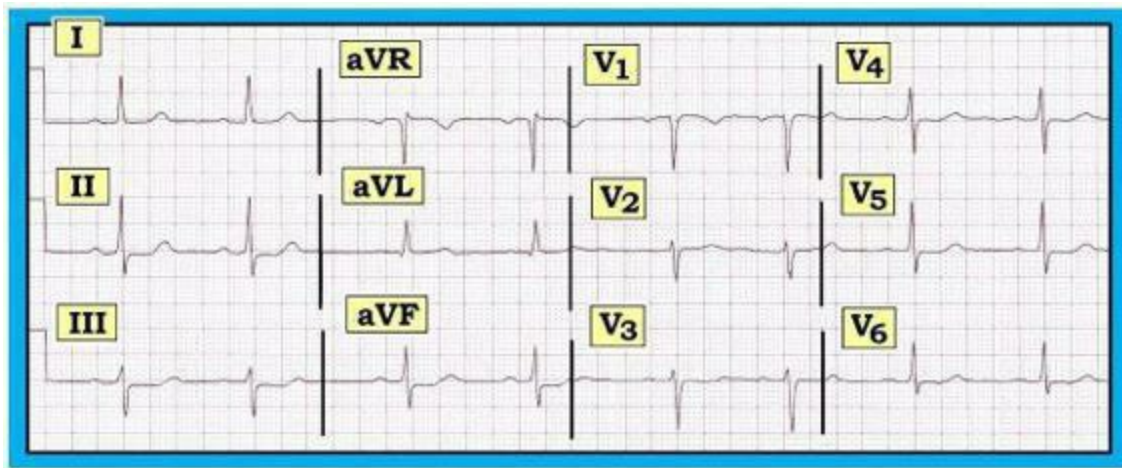


Figure 09.30-1: ECG obtained from an adult with *atypical* chest discomfort. Would you interpret this ECG as a “normal” tracing?

Answer to Figure 09.30-1: The rhythm is sinus bradycardia and arrhythmia (*heart rate ~60/minute, or a bit below this*). The PR, QRS and QT intervals are all normal — as is the axis (*which is about +30 degrees*). There is no chamber enlargement.

- **Q-R-S-T Changes:** A small and narrow q wave is seen in lead aVL. Transition is slightly delayed (*the R wave becomes taller than the S wave is deep between V4-to-V5*). The most remarkable finding on this ECG is **ST segment flattening with slight ST depression in multiple leads**.
- The *amount* of actual ST segment depression on this tracing is minimal (*no more than 1mm in the inferior leads*) — yet there is *no denying* that ST depression is present (*See blow-up inserts in the inferior leads in Figure 09.30-2*).
- There is *no* ST depression at all in leads I and V2-through-V6 (*Figure 09.30-2*). That said — ST-T waves are not normal in these leads. Instead — there is *subtle-but-real* **ST segment straightening** that resembles the picture in **Panel B** of *Figure 09.29-1*.

BOTTOM Line: The ECG in **Figure 09.30-2** is *not* normal. Instead — there is diffuse **nonspecific ST flattening** and **slight ST depression**. *These changes are subtle but real*. Clinical correlation is essential for knowing how to interpret this ECG finding. This patient may have coronary disease — possibly even *severe* coronary disease. On the other hand — these changes are *not* acute and they could be due in part or in combination to *any* of the *other* potential causes of ST depression (*drug effect, electrolyte disorder, hyperventilation, acutely ill patient, etc.*). We simply *cannot* tell on the basis of this single ECG.

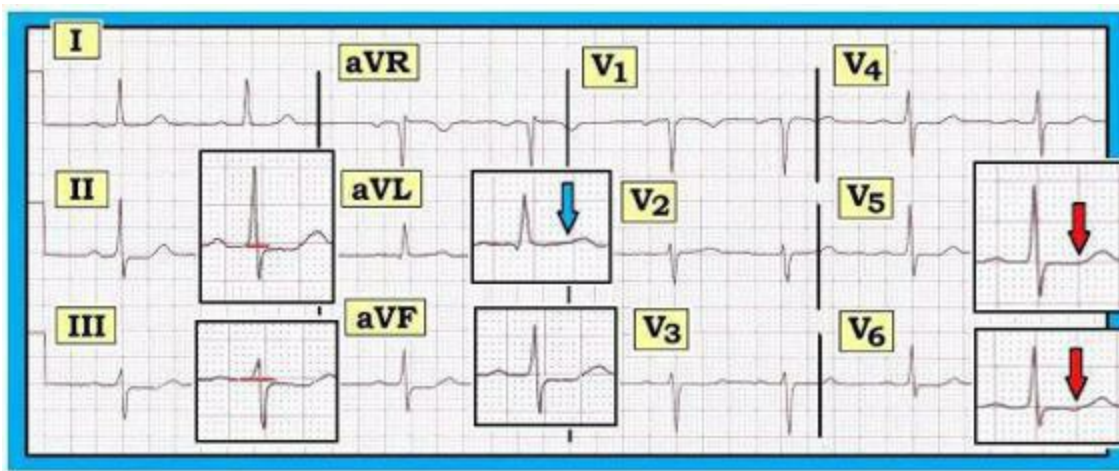


Figure 09.30-2: Reproduction of the ECG in [Figure 09.30-1](#), with *blow-up* inserts illustrating *subtle* ST-T wave abnormalities. Note that there is ST depression (*of ~1mm*) in the inferior leads. There is also ST segment flattening (*straightening*) but *no* depression in leads I and V2-through-V6 (*red arrows in blow-up inserts in V5, V6*). Although T wave amplitude in lead aVL is reduced — note that gradual transition from ST segment-to-T-wave is preserved in this lead (*blue arrow*) — compared to clear *straightening* of the ST segment in leads V5, V6 (*red arrows*). This is not a “normal” ECG (See *text*).

aVR ➡ **How to use Lead aVR**

In years past — We virtually *ignored* **lead aVR**. *No longer!* This once forgotten *right-sided* lead is now appreciated for a series of ***Pearls*** that it may provide in the diagnosis of a number of *special* but *important* situations. These include identification (*or at least suggestion*) of the following:

- **LMCA** (*Left Main Coronary Artery*) **disease** or **occlusion** (*Section 09.40*).
- **Proximal LAD** (*Left Anterior Descending*) **occlusion** (*Section 09.40*).
- **Severe Coronary Artery Disease** (*Section 09.40*).
- **Lead Misplacement/Dextrocardia** (*Section 09.32*).
- **Acute Pulmonary Embolus** (*Section 09.33*).
- **Acute Pericarditis** (*Section 09.34*).
- **Atrial Infarction** (*Section 09.35*).
- **Confirmation of Ventricular Tachycardia** (*Section 09.37*).
- **Diagnosis of some SVT Rhythms** (*Section 09.36*).
- **TCA** (*TriCyclic Antidepressant*) **Overdose** (*Section 09.38*).
- **Takotsubo Syndrome** (*Section 09.39*).

Our goal in this last part of Section 09 — is to illustrate how use of **lead aVR** may serve as a *valuable* adjunct to ECG interpretation. Some of the tracings shown here have already been encountered. Others will be encountered again when addressing future topics (*such as acute pericarditis*). The **common “theme”** — is that by *actively* incorporating assessment of **lead aVR** into your systematic approach, diagnostic accuracy will improve for a surprising number of clinical conditions!

- **NOTE:** Much of what follows represents ***advanced concepts*** that less experienced interpreters need *not* necessarily concern themselves with *unless* looking to learn more. We emphasize that **lead aVR** is *not* essential for the basics of ECG interpretation. That said — awareness of the insight that using lead aVR may provide will help advance your ECG interpretation ability to the *next* level.

09.32 – Lead aVR: Recognizing Lead Misplacement/Dextrocardia

We devoted Section 03 to discussion of lead derivation and recognition of potential technical errors in lead placement. The remote, *right-sided* location of *augmented* **lead aVR** (*looking down at the heart from perspective of the right shoulder*) — provides a unique viewpoint that allows rapid recognition when something is amiss (*Figure 09.32-1*).

- The ECG waveform in **lead aVR** is derived from the *difference* in electrical potential between that recorded from the RA (*right arm*) electrode — *minus* the potential recorded from a central

reference point.

- As shown in **Figure 09.32-1** — the remote *right-sided* and *superior* location of lead **aVR** typically records a complex that manifests **global negativity** (of the *P* wave, *QRS* and *T* wave). While *other* conditions exist that may produce a complex with significant R wave positivity in lead aVR (ie, *RVH*, *COPD*, *conduction defects*, etc.) — the **possibility** of **technical error** should *at least* be contemplated when the QRS in lead aVR is predominantly positive.

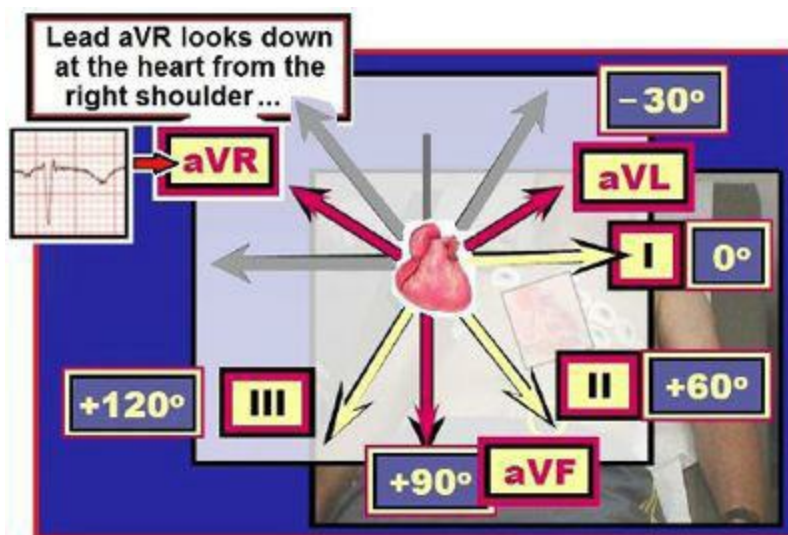


Figure 09.32-1: Hexaxial lead system (reproduced from Figure 03.4-1). The remote *superior* and *right-sided* perspective of lead **aVR** (looking down at the heart from the right shoulder) — typically results in an ECG appearance of **global negativity** (negative *P* wave, *QRS* and *T* wave) in lead aVR, since predominant electrical activity is seen as moving away from this remote lead. **NOTE:** Exceptions exist (*the P wave and T wave in aVR are not always negative*) — but in general, the finding of a predominantly *positive* narrow QRS complex in lead aVR should prompt one to contemplate RVH, COPD or some technical error/dextrocardia.

Consider the ECG in **Figure 09.32-2**:

- What is *unusual* about lead aVR?
- What is *unusual* about lead I?
- Why does this tracing *not* represent dextrocardia?

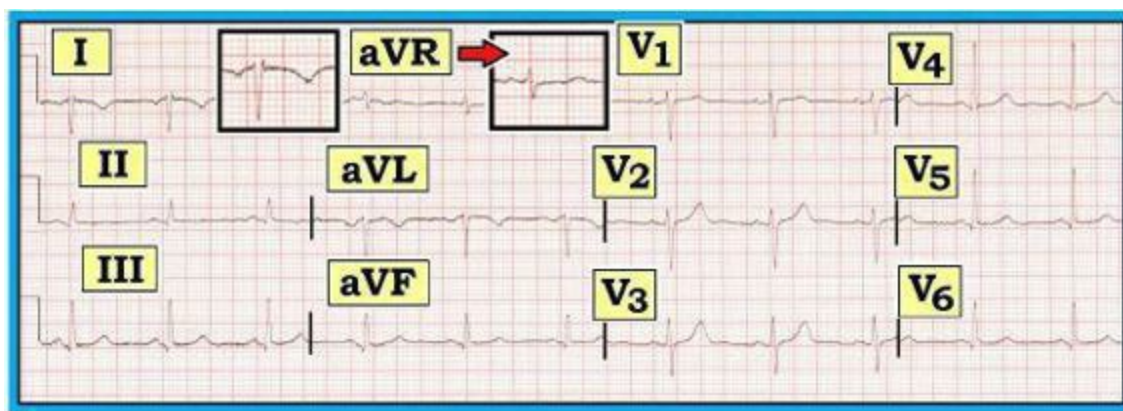


Figure 09.32-2: What are the *unusual* findings in leads I and aVR of this tracing (ECG reproduced from Figure 03.16-1). Why does this tracing not represent dextrocardia? (See text).

Answer to Figure 09.32-2: Two findings should *immediately* capture your eye in assessing this 12-lead tracing: **i)** There is **global negativity** in **lead I** (the P wave, QRS and T wave are all negative); **and ii)** **Lead aVR** does not look as we normally expect it to look.

- Given the **left-sided location** of **lead I** — there should virtually never normally be global negativity (of P wave, QRS, T wave) in this lead. *Think dextrocardia or limb lead reversal!*
- Support that something is *amiss* is forthcoming from **assessment** of **lead aVR**. Note that both the P wave and T wave are positive in aVR. In addition — there is an initial *positive* r wave deflection, instead of the QS or Qr complex that is most commonly seen in this lead.
- Whatever might be wrong in **Figure 09.32-2** is not a result of dextrocardia. We say this because R wave progression is normal — with transition (where the R wave becomes taller than the S wave is deep) occurring between lead V3-to-V4, which is normal. R wave progression should be *reversed* IF the heart was situated in the *right* side of the thorax.

The possibility of **limb lead reversal** is easily settled by **repeat ECG** after verifying lead position (**Figure 09.32-3**). With limb leads now *correctly* placed — Note the following:

- **Lead I** in **Fig. 09.32-3** now manifests a *positive* P wave, QRS complex and T wave, as it normally does.
- **Lead aVR** *now* shows **global negativity** (of P wave, QRS and T wave) — as it most commonly does when lead placement is normal.
- **R wave progression** is **unchanged** compared to **Figure 09.32-2**. Since the technical error only involved reversal of 2 *limb* leads (*mixing up of the right and left arm electrodes*) — there was *no* alteration of precordial lead R wave progression.

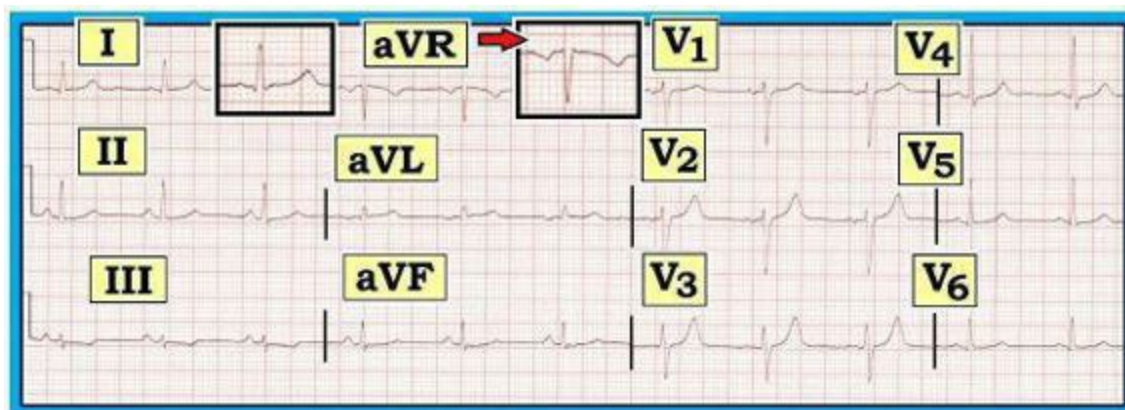


Figure 09.32-3: The ECG initially recorded in **Figure 09.32-2** has been repeated after *verifying* correct limb lead placement. Note that the R wave in lead I is now *positive* — and global negativity has been restored to lead aVR — but that R wave progression is *unchanged*. This confirms **limb lead reversal** as the cause of the *unusual* ECG findings that were seen in **Figure 09.32-2** (See text).

BOTTOM Line: — Technical errors will *definitely* be seen from time to time. Dextrocardia is rare — but important to recognize when it does occur. **Full Review** of this subject is covered in **Section 03**. Suffice it to say here — that attention to **lead aVR** is an easy way to facilitate recognizing *many* of the common technical mishaps (**Figure 09.32-4**).

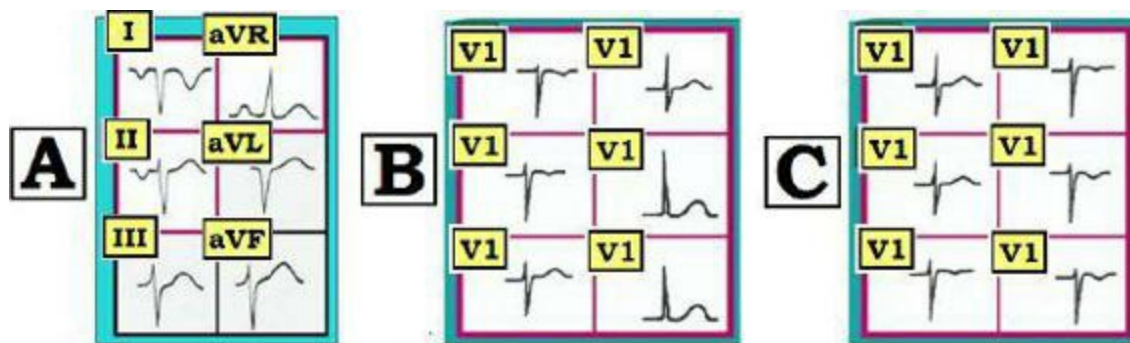


Figure 09.32-4: Limb lead appearance in **Panel A** should *immediately* suggest something is amiss. This is because: **i)** there is *global negativity* in **lead I**, which is virtually *never* seen under normal circumstances; **ii)** **Lead aVR** shows **global positivity** (which is also virtually *never* seen normally); and **iii)** the P wave is *not* upright in **lead II** (which means either a non-sinus rhythm; dextrocardia, or lead reversal). IF the precordial lead sequence for the limb leads in **Panel A** looked like **Panel B** (which shows normal R wave progression) — We would suspect left and right arm limb lead reversal. But IF the precordial lead sequence looked like **Panel C** — the reverse R wave progression in C would strongly suggest dextrocardia (See Section 03.11 for more details).

09.33 – Lead aVR: in Acute Pulmonary Embolus

Interpret the ECG shown in **Figure 09.33-1** — obtained from a patient who presented with syncope, shock and acute hypoxemia.

- What *clinical* condition is suggested by this history and the *combination* of ECG findings seen in **Figure 09.33-1**?
- Does this clinical diagnosis account for the **ST elevation** seen in **lead aVR**?

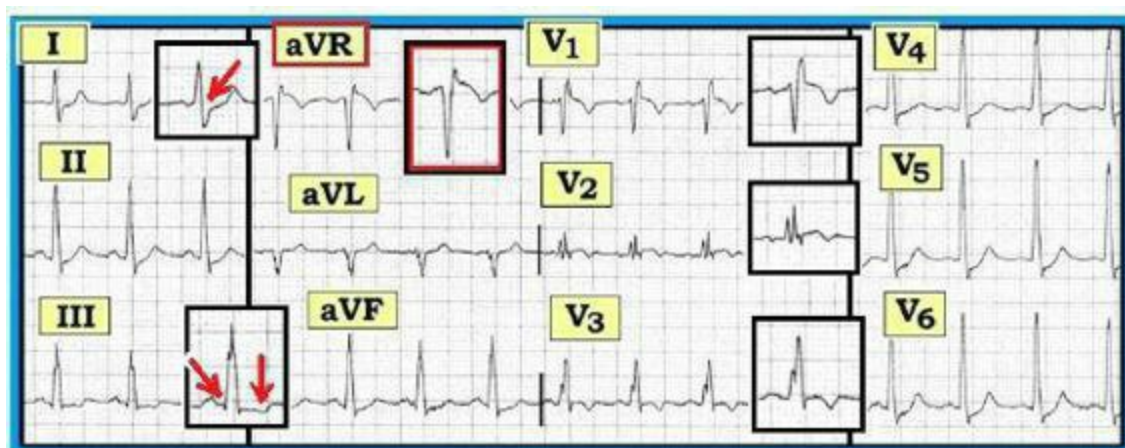


Figure 09.33-1: ECG obtained from a patient who presented with syncope, shock and acute hypoxemia (reproduced from **Figure 08.32-1**). What **clinical diagnosis** would account for *all* of the ECG findings seen in this tracing (including ST elevation in lead aVR)?

Answer to Figure 09.33-1: As discussed in detail in Sections 08.34 *through* 08.38 — the history and combination of ECG findings in Figure 09.33-1 should strongly suggest the diagnosis of submassive *acute PE* (*Pulmonary Embolism*). ECG findings supportive of this diagnosis include:

- Sinus tachycardia.
- RBBB (*Right Bundle Branch Block*).
- Diffuse ST-T wave abnormalities consistent with RV “strain”.
- S1Q3T3 pattern (*Section 08.36*).
- **ST elevation** that is essentially *limited* to **lead aVR** and lead V1.

We emphasize — that one can *not* rule out the possibility of acute *anterior* MI from the ECG shown in Figure 09.33-1. That said — virtually *all* facets of the history and this ECG can be explained by one *unifying* diagnosis = a hemodynamically *significant large acute PE*.

- Anterior (*as well as inferior*) ST-T wave abnormalities as seen in Figure 09.33-1 are consistent with RV “strain”.
- Very few conditions produce **ST elevation** that is *most marked* in **lead aVR** — but minimal to *nonexistent* elsewhere, with possible exception of *right-sided* lead V1. One of these conditions is *acute PE*, as a result of acute *right* heart “strain” (*as seen in this case*). This patient was found to have a large saddle embolus.

09.34 – Lead aVR: in Acute Pericarditis

Acute pericarditis is discussed in detail in Section 12. The hallmark of the ECG diagnosis of *acute pericarditis* is *generalized ST elevation* that occurs in a clinical setting consistent with this diagnosis. *Reciprocal* ST depression and large Q waves are *absent*. ST elevation typically manifests an *upward* concavity (“*smiley*”-shape) in most leads — with possible exception of *right-sided* leads (*leads aVR, V1 and sometimes lead III*) that may show T wave inversion.

- In addition to *generalized* ST elevation — **PR segment depression** in at least *several* leads (*below the TP baseline*) is another helpful clue in support of the ECG diagnosis of acute pericarditis. PR depression is well seen in Figure 09.34-1 (*red arrows*).
- The value of using **lead aVR** when contemplating a diagnosis of acute pericarditis — is that this lead often manifests **PR segment elevation** (*blue arrow in Figure 09.34-1*). As will be emphasized in Section 12 — PR segment depression is far from definitive for confirming acute pericarditis. Nevertheless, recognition of **PR depression** in *several* leads in association with **PR elevation** in **lead aVR** — *together with*: i) *generalized concave-up* ST elevation in the *absence* of reciprocal ST depression; and ii) a clinical setting *consistent* with acute pericarditis — go a long way toward increasing our comfort level in the likelihood of this diagnosis.

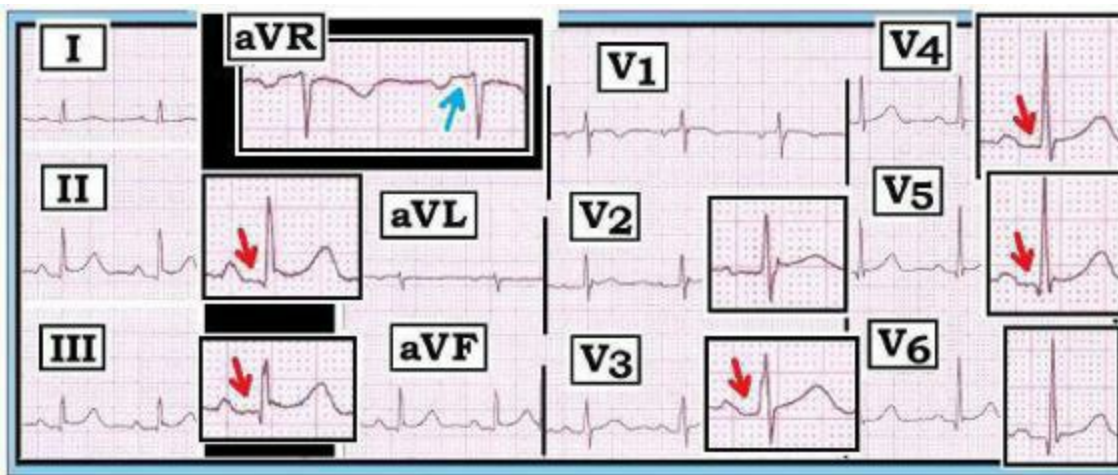


Figure 09.34-1: ECG obtained from a 35-year old man with *atypical* chest pain. The ECG diagnosis of **acute pericarditis** is suggested by: **i)** *generalized* concave-up ST elevation; **ii)** *absence* of reciprocal ST depression; **iii)** *absence* of large Q waves; **iv)** PR depression in a number of leads (*red arrows*); **and v)** **PR elevation** in lead **aVR** (*blue arrow*). Acute pericarditis is discussed in detail in Section 12.

09.35 – Lead aVR: in Atrial Infarction

Atrial infarction is rare. ECG findings are subtle and often overlooked. As a result — most cases of atrial infarction are only diagnosed post-mortem on autopsy study. *Realistically Speaking* — being able to diagnose acute atrial infarction is unlikely to alter management. As a result — one is probably *none the worse* IF you *ignore* routinely looking for signs of atrial infarction (*as many clinicians seem to do*). That said — **recognition** of **acute atrial infarction** may explain *abrupt* onset of *supraventricular* arrhythmias that are prone to occur in association with *acute* MI. We therefore include this subject as an *advanced* topic. **Consider** the possibility of **acute atrial infarction** in the following clinical setting(s) — and in the presence of the following **ECG signs**:

- There is an **ongoing acute STEMI** (*ST Elevation Myocardial Infarction*) that appears to be large (*as suggested by significant ST elevation and marked reciprocal ST depression*).
- Proximal occlusion of a major coronary artery is suggested by the ECG or acute catheterization picture (*the SA nodal artery is supplied by the Right Coronary Artery in ~60% of cases — with the circumflex accounting for most of the rest, though anatomic variations are possible*). By far, **acute inferior MI** — is the most common location for *acute* MI associated with atrial infarction. Whether this is the result of more likely origin for the SA nodal artery from the RCA — or due to the much higher oxygen content of left atrial blood is uncertain. There is often *associated* ECG evidence of **acute posterior** and **RV** (*Right Ventricular*) **involvement** — supporting the likelihood of *proximal* RCA occlusion (**Figure 09.35-1**).
- There is **abrupt onset** of **AFib** or *other* atrial tachyarrhythmia in association with *acute* STEMI. Less commonly there may be PR interval prolongation, sinus pauses or sinus arrest. Abrupt loss of the atrial kick in a patient with a large acute MI may result in sudden cardiac decompensation (*from acute heart failure*).
- In addition to *acute* STEMI (*usually from inferior MI*) — there are **acute PTa deviations** (*elevation or depression of the T wave component of the preceding P wave, during which time atrial repolarization takes place = “PTa” wave*). These PTa segment deviations may vary in lead location, depending on *which* atrium and *what part* of the atria is infarcting (*left or right*).

atrium; atrial free wall or appendage). The ECG picture in *schematic* **Figure 09.35-1** — is typical for the changes one might expect with acute right atrial infarction.

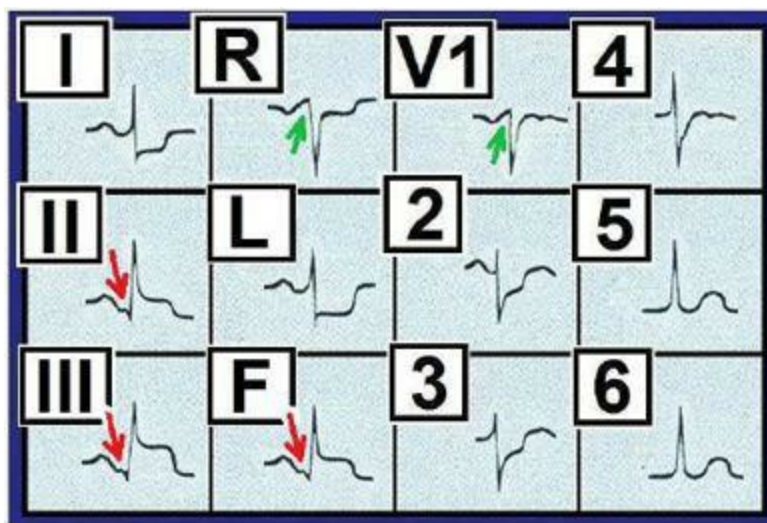


Figure 09.35-1: *Schematic* ECG illustrating expected findings from acute right atrial infarction. Note: **i)** underlying acute *infero-postero* MI; **ii)** PTa segment depression in the *inferior* leads (red arrows) — and perhaps also in V2; **and iii)** PTa elevation in lead aVR and lead V1 (green arrows). *Beyond-the-Core:* Proximal RCA occlusion is suggested in this schematic tracing by the finding of *more* ST elevation in lead III than lead II and by *marked* ST depression in lead aVL. Acute RV involvement is suggested by the relatively flat ST segment in lead V1 in the face of *marked* ST depression in V2. *Right-sided* leads could confirm acute RV involvement.

Now look at the ECG in **Figure 09.35-2** — obtained from a patient with chest pain. There is obvious acute *inferior* STEMI — which is most probably from acute RCA occlusion (*marked ST elevation in lead III > II*; *marked ST depression in lead aVL*). ST depression in leads V1 and V2 suggest associated *posterior* involvement.

- Do you *also* see ECG signs suggestive of **acute atrial infarction** in **Figure 09.35-2**? *Wouldn't it be easy to overlook this?*

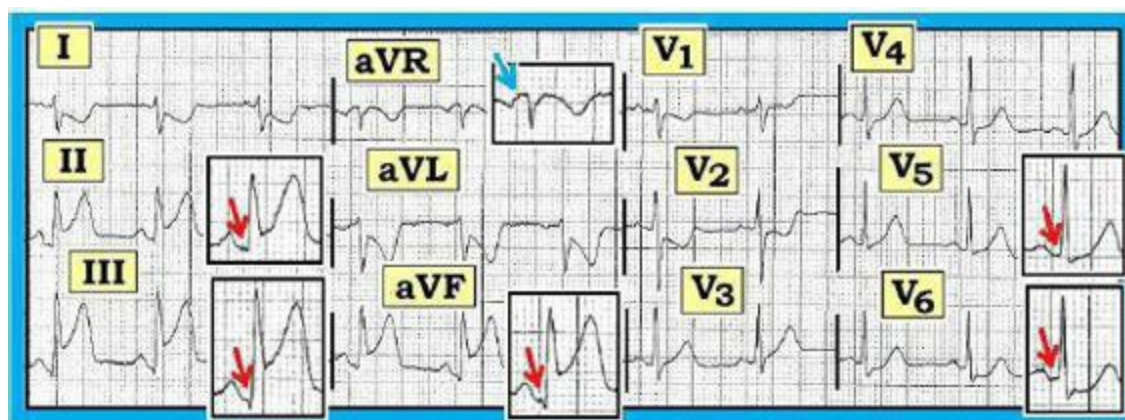


Figure 09.35-2: Acute *infero-postero* STEMI. In addition — there is ECG evidence of **acute atrial infarction**. This is suggested by PTa depression in leads II,III,aVF and V5,V6 (red arrows) — *plus*

PTa elevation in lead aVR (*blue arrow*).

Bottom Line: Atrial infarction is rare. Even when present, this diagnosis is rarely made by ECG while the patient is still alive — as ECG signs are often extremely subtle (Figure 09.35-2). Nevertheless — attention to the clinical setting and recognition of PTa segment deviations (*PR segment elevation or depression*) may suggest the diagnosis and explain certain potential complications of acute myocardial infarction. This is an advanced topic that clearly extends “*beyond-the-core*”.

- Beyond-the-Core: Note that *both* acute pericarditis (Figure 09.34-1) — *and* atrial infarction (Figure 09.35-2) — are examples of relatively uncommon *clinical* conditions that may result in **PR depression** in several leads with **PR elevation** in lead aVR. Clinical circumstances and the *overall* ECG picture allow easy distinction between these 2 conditions.

Reference: Shakir DK, Arafa SOE: *Right Atrial Infarction, Atrial Arrhythmia and Inferior MI from a Missed Triad: Case Report & Review of the Literature*. Can J Cardiol 23:995-997, 2007.

09.36 – Lead aVR: in Supraventricular Arrhythmias

Among the most helpful uses of lead aVR is in the interpretation of cardiac arrhythmias. **Lead aVR** may be *beneficial* in **2 principal ways**: **i)** in detecting subtle *atrial* activity in certain *supraventricular* tachyarrhythmias (*Section 09.36*); and **ii)** in making a definitive diagnosis of VT (*Section 09.37*).

- In general — the *best* lead to look at for assessment of atrial activity is **lead II**. This is because IF the **P wave** is *upright and* conducting in lead II (ie, *the PR interval is constant*) — then there is **sinus rhythm**. In addition — P wave amplitude is often greatest in lead II, which makes P waves easy to see under normal circumstances.
- The *next-best* lead to look at for assessment of atrial activity is **lead V1**. This is because this *right-sided* lead conveniently provides an *anatomically* close electrical perspective of normal atrial activity as the atria are depolarized.
- Clinically — atrial activity is *not* always clearly evident in leads II and V1. This is especially true for certain *regular* SVT (*SupraVentricular Tachycardia*) rhythms — in which the rapid rate results in a *shortening* of the R-R interval. This may provide a “cover” (*the preceding T wave*) — within which atrial activity may be hiding. The unique electrical vantage point of **lead aVR** may occasionally help to overcome this problem by providing a clue to underlying atrial activity that may *not* be as readily evident in *other* leads (**Figure 09.36-1**).

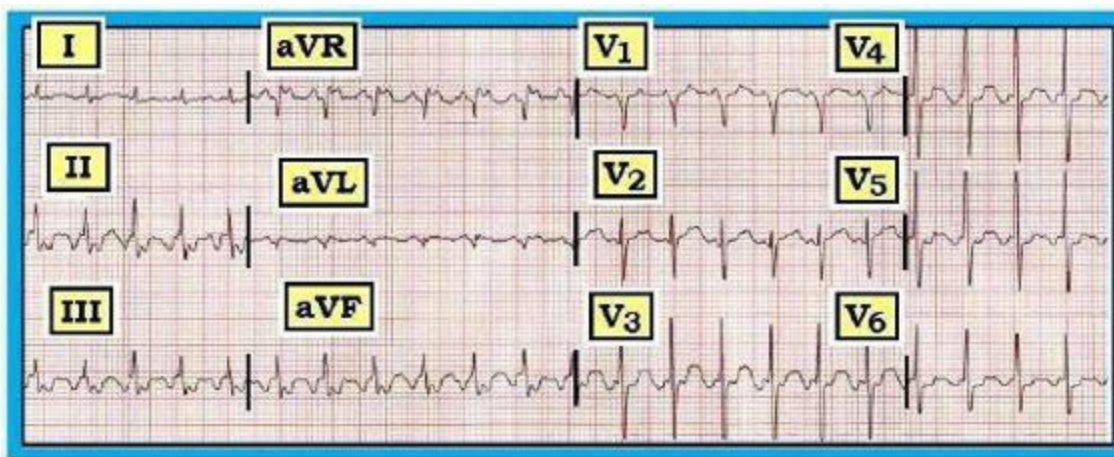


Figure 09.36-1: 12-lead ECG demonstrating a **regular SVT rhythm** at ~150/minute *without* normal atrial activity (ie, *no upright P wave in lead II*). **Question:** Is there evidence of **atrial activity** in **other leads**? (See text).

Answer to Figure 09.36-1: The rhythm is a **regular SVT** at ~150/minute — but *without* normal atrial activity (since lead II does not show an upright P wave). The principal **differential diagnosis** consists of 3 entities: **i)** Sinus tachycardia; **ii)** PSVT; and **iii)** Atrial flutter. As discussed in Section 02.19 — looking at **additional leads** in the hope of identifying **atrial activity** may be insightful.

- **PEARL:** Use of **calipers** facilitates the process. Setting one's calipers at *precisely* half the R-R interval — allows you to walk out atrial activity in several leads (**Figure 09.36-2**). This **confirms** the diagnosis of **AFlutter** with 2:1 AV conduction (*atrial rate = 300/minute; ventricular rate = 150/minute*).
- Note how helpful looking at **lead aVR** is in **Figure 09.36-2** — for increasing our level of comfort that 2:1 AV conduction is *truly* present. Clear demonstration of 2:1 AV conduction in multiple leads (*red and blue arrows*) may obviate diagnostic need for performing a vagal maneuver.

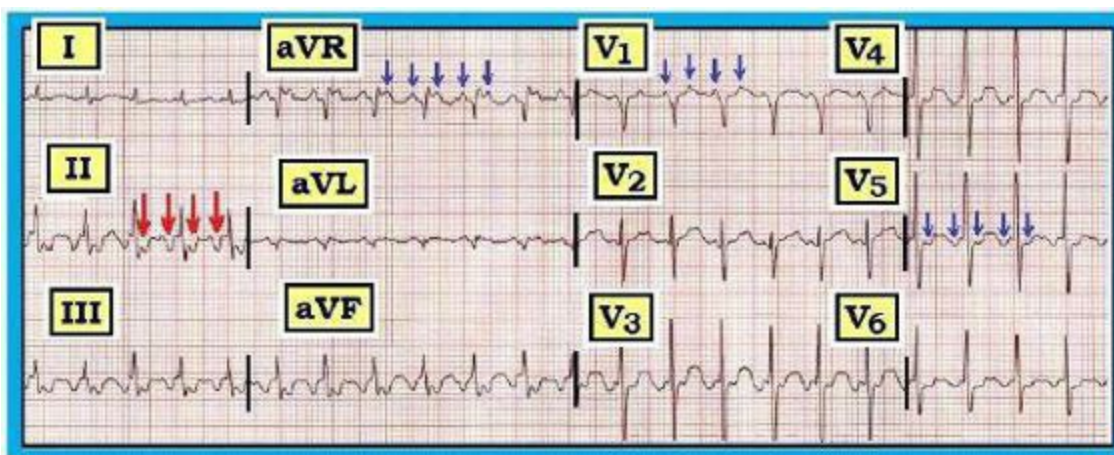


Figure 09.36-2: Arrows have been added to **Figure 09.36-1** to facilitate recognition of atrial activity. Note that 2 *evenly-spaced* negative deflections are present for each QRS complex in lead II (*red arrows*). Regular flutter waves are also well seen in **lead aVR**, as well as in selected other leads (*blue arrows*).

Retrograde Atrial Activity: Another way in which *additional* leads may facilitate diagnosis of *regular* SVT rhythms — is in identifying **retrograde atrial activity**. Our 2 “*favorite*” leads to look for *retrograde* P waves in — are **lead aVR** and **lead V1** (Figure 09.36-3).

- As emphasized in Section 02.29 and Section 02.30 — **PSVT** is a **reentry tachycardia** that usually involves at least *some* portion of the AV node. Confirmation that **reentry** is the mechanism of a *regular* SVT rhythm when normal P waves are absent — is forthcoming from identifying *retrograde* atrial activity during the tachycardia.
- **Retrograde P waves** during PSVT are often subtle, if evident at all. They may be nothing more than a *tiny* notch seen in the *terminal* portion of the QRS in one or more of the *inferior* leads (*red arrows in Figure 09.36-3*).
- Support that such notching is *real* and a reflection of *retrograde* atrial activity — is forthcoming from identifying retrograde P waves in *other* leads. While **retrograde P waves** are *negative* when seen in lead II or other inferior leads — they are generally **positive** when seen in **lead V1** and/or **lead aVR** (*blue arrows in Figure 09.36-3*).

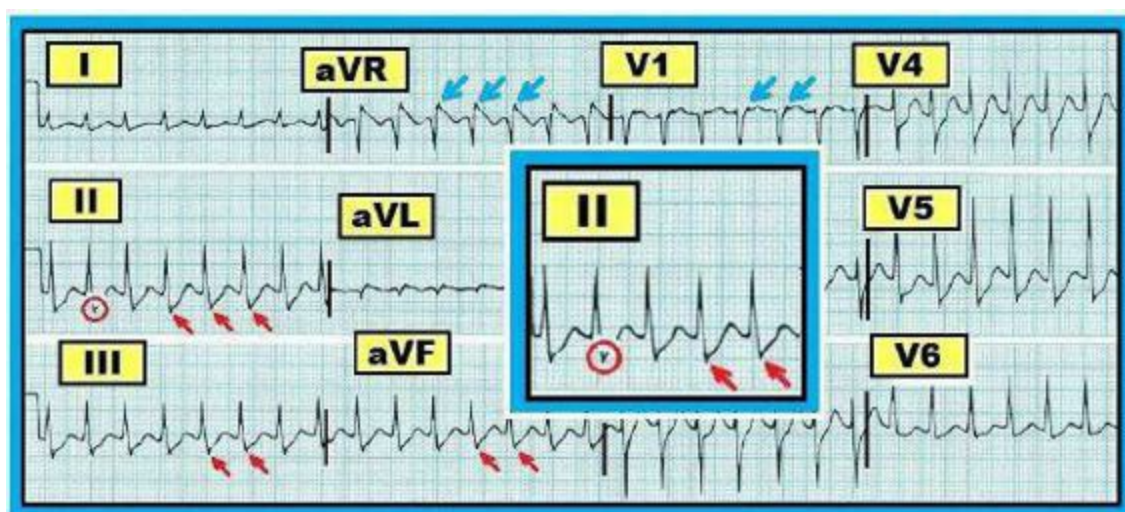


Figure 09.36-3: *Regular* SVT rhythm *without* normal P waves at a rate of ~180/minute (*reproduced from Figure 02.30-1*). Retrograde atrial activity is suggested by subtle *negative* notching in the terminal part of the QRS in each of the inferior leads (*red arrows — which are best seen in the lead II blow-up in the center of the tracing*). Support that such notching is *real* and reflects *retrograde* P waves from a *reentry* mechanism — is forthcoming from the “pseudo-r-prime” (*positive terminal notching*) that is seen in the QRS complex in *both* leads aVR and V1 (*blue arrows*). This **confirms reentry** and the diagnosis of **PSVT** as the mechanism of the arrhythmia (*See text*).

09.37 – Lead aVR: for Definitive Diagnosis of VT

In Sections 02.47 through 02.51 — We emphasized the importance of *always* assuming a **regular WCT** (Wide-Complex Tachycardia) **rhythm** was **VT** (Ventricular Tachycardia) until proven otherwise. This is because: **i)** VT is by far (>80-90% of the time) the most common cause of a *regular* WCT rhythm when sinus P waves are *not* evident; and **ii)** VT is the most serious cause of a *regular* WCT. That said — *Wouldn't it be nice* to be able to *increase* our diagnostic *certainty* that a *regular* WCT was VT beyond this 80-90% likelihood probability?

- Consider the **regular WCT rhythm** shown in **Figure 09.37-1** — obtained from an adult with a history of heart disease. *How certain* are you that the rhythm is VT?

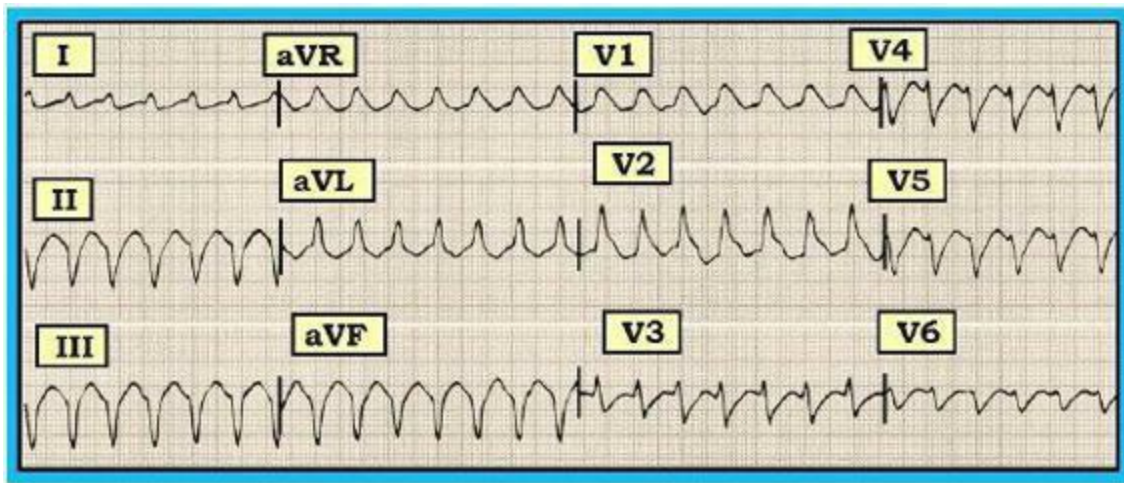


Figure 09.37-1: Regular WCT rhythm obtained from a patient with heart disease. *How certain* are you that the rhythm is VT? (See text).

Answer to Figure 09.37-1: The rhythm is a **regular WCT** at **~180/minute**. There is no sign of atrial activity. VT should be assumed until proven otherwise (**LIST #1** — from **Figure 02.47-1**).

- In **Section 02.50** — We emphasized how use of **3 Simple Rules** can increase diagnostic likelihood *beyond* the 90% probability level predicted by the presence of any *regular* WCT rhythm in an adult with heart disease. Applying these **3 Simple Rules** from Section 02.50 to the rhythm in **Figure 09.37-1** — Note that there is: **i)** *extreme* axis deviation (*entirely negative QRS in lead aVF*); **ii)** amorphous = “ugly” and very wide QRS morphology (*the QRS is well over 0.16 second in duration*); and **iii)** an almost *entirely* negative QRS complex in lead V6. We estimate that these ECG findings during tachycardia increase VT likelihood to ~98%.
- The *additional* finding of an **entirely upright (monophasic) QRS complex** in lead **aVR** during this WCT rhythm increases **diagnostic certainty** of VT to **virtually 100%**! This is because the only way a monophasic (*entirely positive*) QRS complex can be seen in lead aVR during WCT — is **IF** the electrical impulse originates from a site in the ventricular apex. *Nothing else other than VT does this (Figure 09.37-2).*
- In contrast — we learn nothing about the etiology of a WCT rhythm **IF** lead aVR is either initially negative — or manifests anything but a large monophasic R wave (*2nd and 3rd examples in Figure 09.37-2*).

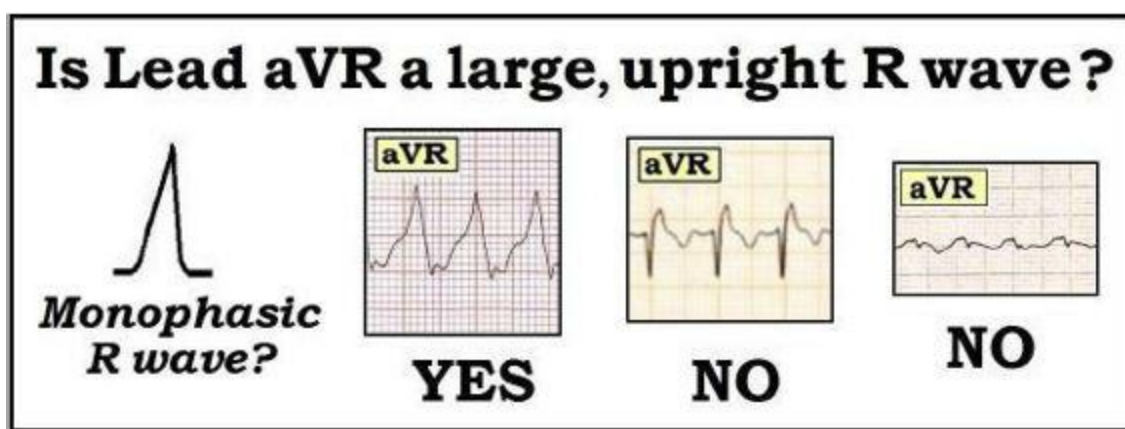


Figure 09.37-2: The finding of a **large monophasic R wave** in lead aVR during a WCT rhythm is **diagnostic** of VT with virtual 100% accuracy. In contrast — *anything but* a large monophasic R wave (2nd and 3rd examples in this figure) is of no diagnostic utility in differentiation (See text).

BOTTOM Line: The criterion of a **monophasic upright R wave** in lead aVR is admittedly insensitive. As a result — this finding will only be seen in a minority of WCT rhythms. Nevertheless, we mention it as a **special use** of lead aVR in the diagnosis of cardiac arrhythmias — because in those few cases when a monophasic R wave is seen in lead aVR (**Figure 09.37-3**) — the diagnosis of VT is *virtually* assured.

- As should be obvious — diagnosis of VT is *overwhelmingly* likely in Figure 09.37-3, even *without* use of lead aVR. That said — once lead aVR appearance is noted, there is *no longer* the *slightest* doubt that the rhythm is VT (*which allows us to focus full effort on treatment*).

Reference: Sasaki K: *A New Simple Algorithm for Diagnosing Wide QRS Complex Tachycardia: Comparison with Brugada, Vereckei and aVR Algorithms*. Circulation 120:S671, 2009.

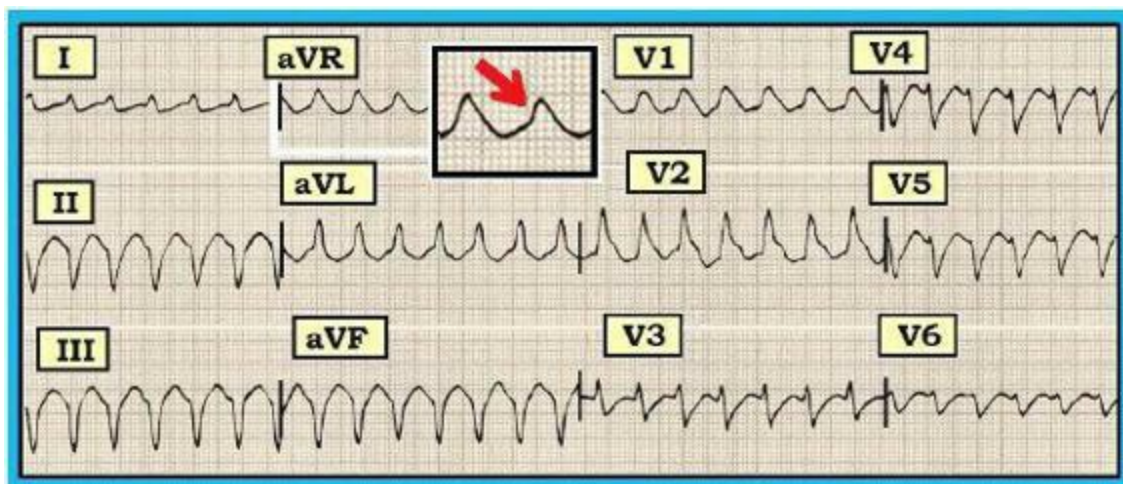


Figure 09.37-3: We reproduce Figure 09.37-1 — with a *blow-up* insert of lead aVR. The presence of a **monophasic R wave** in lead aVR makes diagnosis of VT a virtual certainty (See text).

09.38 – Lead aVR: in TCA Overdose

Severe TCA (TriCyclic Antidepressant) Overdose — may produce characteristic ECG manifestations that are in part defined by **lead aVR**. Although far less commonly encountered than in the past — *severe* TCA poisoning is still occasionally seen. It is well to remember that overdose of

other medications (ie, *quinidine; procainamide; flecainide*) may produce a similar ECG picture of sodium-channel blockade. The principal **adverse effects** of **severe TCA overdose** include: **i**) Neurotoxicity (*seizures; delirium*); and **ii**) Cardiovascular toxicity (*life-threatening ventricular tachydysrhythmias; myocardial depression; hypotension; preterminal bradycardia*). The ECG picture may be unique (Figure 09.38-1) — and is recognized by the following:

- **Sinus Tachycardia** — is almost always seen with severe TCA ingestion until very late in the course (*ultimately there may be preterminal bradycardia*).
- **QRS prolongation** — to at least 0.10 second. Rather than the QT — it is QRS prolongation that best predicts severity of the overdose. Potentially lethal ventricular arrhythmias are more likely once QRS duration exceeds 0.16 second. Of interest — QRS prolongation especially affects the *terminal* portion of the QRS, such that the initial part of the QRS is often not overly abnormal.
- **RAD** (*Right Axis Deviation*) — which manifests as: **i**) a wide terminal S wave in lead I; and **ii**) a **prominent R'** (*>3mm tall*) in lead **aVR**, that is often taller than the S wave is deep in this lead.
- **Predisposition to potentially lethal VT** (*Ventricular Tachycardia*).
- **Other ECG findings** that may be seen in severe TCA overdose include: **i**) QT prolongation; **ii**) PR interval prolongation; **iii**) RBBB; **iv**) a transient Brugada pattern in anterior precordial leads.

Clinical NOTE: Given that ECG signs of **severe TCA toxicity** include: **i**) *marked* QRS widening and **ii**) PR as well as QT prolongation — it may at times be difficult (*if not impossible*) to distinguish between: **i**) sinus tachycardia with a wide QRS and 1st-degree AV block (*with P waves hidden within tall T waves made prominent by QT prolongation*); vs **ii**) VT.

- Fortunately — clinical distinction between these 2 forms of *wide-complex* tachycardia (*VT vs sinus tach*) is not essential for appropriate *initial* management because: **i**) **Sodium Bicarbonate** is recommended as the drug of choice regardless of whether the wide rhythm is sinus tachycardia or VT; and **ii**) antiarrhythmic drugs such as procainamide, amiodarone, sotalol or flecainide are all contraindicated for treatment of VT with TCA overdose (*because these agents may all aggravate myocardial depression, hypotension, and conduction defects*).
- **Bottom Line:** Clinical context (ie, *that the patient overdosed on a TCA*) — *prior* ECGs on the patient — and *serial* tracings may sometimes be needed in order to become comfortable distinguishing between sinus tachycardia with severe TCA poisoning vs VT (**Figure 09.38-1**).

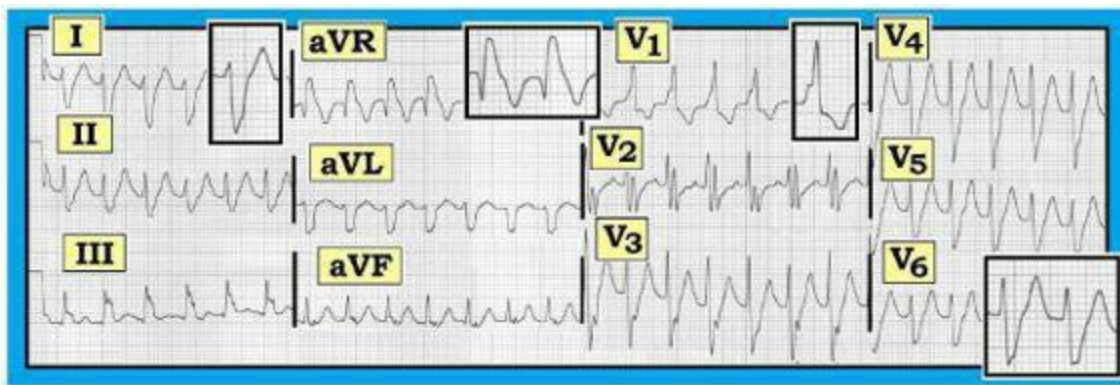


Figure 09.38-1: ECG obtained from a young adult who presented to the ED after TCA overdose. Characteristic ECG signs of **severe TCA toxicity** are present. These include: **i)** Tachycardia; **ii)** Marked QRS widening (to at least 0.16 second) — with QRS widening primarily affecting the terminal portion of the QRS complex; **iii)** RAD — as manifest by a wide terminal S wave in lead I as well as by a **very tall R' component** in lead aVR (well over 3mm in amplitude); **and iv)** RBBB. **NOTE:** We are *not* certain IF the rhythm in Figure 09.38-1 represents VT (marked QRS widening; marked right axis; absence of normal atrial activity in lead II; predominant S wave in lead V6) — or a supraventricular tachycardia with marked QRS widening from severe TCA toxicity (it looks as if P waves may be present in lead V2). In the context of severe TCA toxicity, it probably does *not* matter — since initial management entails Sodium Bicarbonate and avoidance of antiarrhythmic drugs regardless of the etiology of the rhythm (See text).

Acknowledgement: My appreciation to Andrew Bowman for allowing me to publish (with slight modification) the ECG in Figure 09.38-1.

References:

- i) Thanacoody HKR, Thomas SHL: *Tricyclic Antidepressant Poisoning*. Toxicol Rev 24:205-214, 2005.
- ii) Life-In-The-Fast-Lane: <http://lifeinthefastlane.com/ecg-library/basics/tca-overdose/> -

09.39 – Lead aVR: in Takotsubo Syndrome

We discuss the syndrome of Takotsubo Cardiomyopathy with acute *apical* “ballooning” (and resultant acute heart failure) in Sections 10.62, 10.63. Many ECG findings are possible with Takotsubo Cardiomyopathy. ECG changes are often *out-of-proportion* to the clinical picture. Among the many ECG findings that may be seen are ST segment elevation in lead aVR.

- See Section 10.61 for more on Takotsubo Cardiomyopathy.

09.40 – Lead aVR: Severe CAD/Left Main Disease

We have found use of **lead aVR** to be extremely helpful in assessing certain more severe forms of CAD (Coronary Artery Disease) — especially with involvement of multiple vessels *and/or* with left main disease. The illustrative examples that follow hopefully convey this key concept. NOTE: Use of lead aVR in this manner is an advanced topic.

- The remote *superior* and *right-sided* electrical viewpoint of **lead aVR** (looking down at the

heart from the right shoulder) — provides a unique vantage point that assesses the **basal** part of the **interventricular septum**. Ischemia of the septum (*as may occur with severe left main disease*) produces a vector that points superiorly — resulting in **ST elevation** in lead **aVR**. There will often be associated ST elevation in lead aVL and ST depression in the inferior leads).

- Similar ECG findings may often be seen with **acute proximal LAD** (Left Anterior Descending) **occlusion**, in which there is involvement of the 1st septal artery branch.
- In contrast to *proximal* LAD occlusion — **more distal LAD occlusion** generally does not involve the same area of the septum. As a result — ST elevation in lead aVR is not seen. Instead — ST elevation is most marked in *anterior* leads (*V2, V3, V4*).
- Assessment of the *clinical* significance of ST elevation in lead aVR is complicated by the fact that this lead provides reciprocal (*mirror-image*) information to one or more lateral leads. Specifically — ST depression in leads II, aVL, V5, V6 may result in some ST elevation in aVR *independent* of septal involvement.

PEARL: Distinction between *left-main* disease vs *proximal* LAD occlusion may be suggested on ECG by the **relative amount** of **ST elevation** seen in lead **aVR** *compared to* lead **V1**.

- Think **Left-Main** disease — when ST elevation in lead aVR > V1.
- Think **proximal LAD** disease/occlusion — when ST elevation in lead V1 > aVR.

NOTE: Distinction should be made between **acute LMCA** (Left Main Coronary Artery) **occlusion** vs **LMCA disease**.

- Most patients with *acute* LMCA occlusion do not survive. As a result — this entity is not often seen and *unlikely* to be appreciated clinically. Rapid deterioration with patient demise due to cardiogenic shock is the usual result unless acute LMCA occlusion can be *immediately* recognized and *immediately* acted on.
- In those *rare* circumstances when *acute* LMCA occlusion is captured on ECG — rather than diffuse ST depression there should be diffuse precordial ST *elevation* in association with ST elevation in lead aVR.

Consider the ECG shown in **Figure 09.40-1** — obtained from an older adult with a history of chest discomfort over time.

- What is the principal abnormality on this ECG? (HINT: Look at the white arrows).
- Clinically — Is CAD (Coronary Artery Disease) likely? If so — Can you comment on the likely *severity* of such CAD?

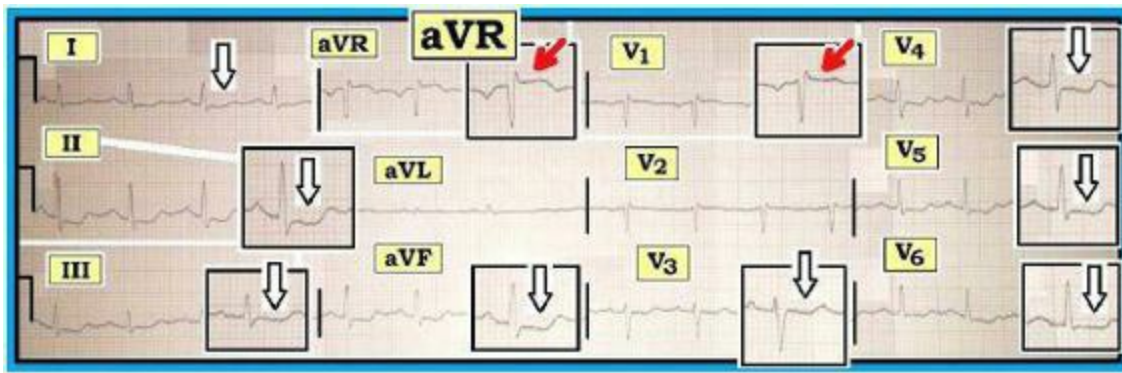


Figure 09.40-1: ECG obtained from an *older* adult with chest pain over time. *Diffuse* ST depression is seen (*white arrows*). In addition, there is ST elevation in leads aVR and V1 (*red arrows*). This pattern suggests *severe* CAD (See text).

Answer to Figure 09.40-1: An important pattern to recognize on ECG — is the finding of *diffuse* ST depression (usually in ≥ 7 -8 leads) in association with ST *elevation* in lead aVR. These findings are seen in Figure 9.40-1:

- Especially when seen in an *older* adult — there is a *high* correlation with this ECG pattern and *severe* CAD (usually 3-vessel — or proximal LAD/left main disease).
- It is difficult to distinguish between *severe* 3-vessel disease vs a proximal LAD or left main lesion on the basis of this ECG alone.
- *Additional* findings of potential concern in Figure 09.40-1 are: **i)** that there is also some ST elevation in lead V1 and **ii)** that *incomplete* RBBB is present (*rSr'* in V1; *tiny-but-present* S waves in leads I, V6).
- Beyond-the-Core: The ST elevation that is seen in lead V1 appears to be *minimal* and is *less* in amount than the ST elevation seen in lead aVR. As a result —the possibility of *left-main* disease should at least be considered. In contrast — *left-main* disease would be far *less* likely IF ST elevation in lead V1 was greater than in lead aVR.
- **BOTTOM Line**: Be aware of the ECG pattern shown in Figure 09.40-1 — in which ST flattening with at least *some* depression is seen in *multiple* leads in association with ST elevation in lead aVR. Especially when seen in an older adult with symptoms consistent with angina — *severe* CAD is likely.

Now consider the ECG shown in Figure 09.40-2 — obtained from an acutely ill patient with severe *new-onset* chest pain. Having just emphasized how clinically *rare* it is to encounter a patient with *acute* LMCA occlusion — We feel this may be one exception. We note the following findings:

- There is sinus tachycardia and QRS widening due to *acute* **bifascicular block** (RBBB/LAHB). Although admittedly difficult to see the low amplitude upright P wave in lead II — it looks like there may *also* be 1st degree AV block.
- There is marked **diffuse ST elevation** (in leads I, aVL; V1-through-V6; and in lead aVR). There is marked **reciprocal ST depression** in inferior leads.
- Small **q waves** have already formed in leads aVL; V1, V2, V3.
- ST elevation is fairly marked (at least 2-3mm) in both lead aVR and in lead V1 (*red arrows*). While we *cannot* be sure if ST elevation is more in aVR or in V1 — this patient's acute

presentation in conjunction with new *bifascicular* block (plus possible 1st degree AV block) and the *dramatic* ST-T wave changes seen here (with **marked ST elevation** in lead aVR) support *high* likelihood of acute *proximal* occlusion (of either the LMCA or proximal LAD).

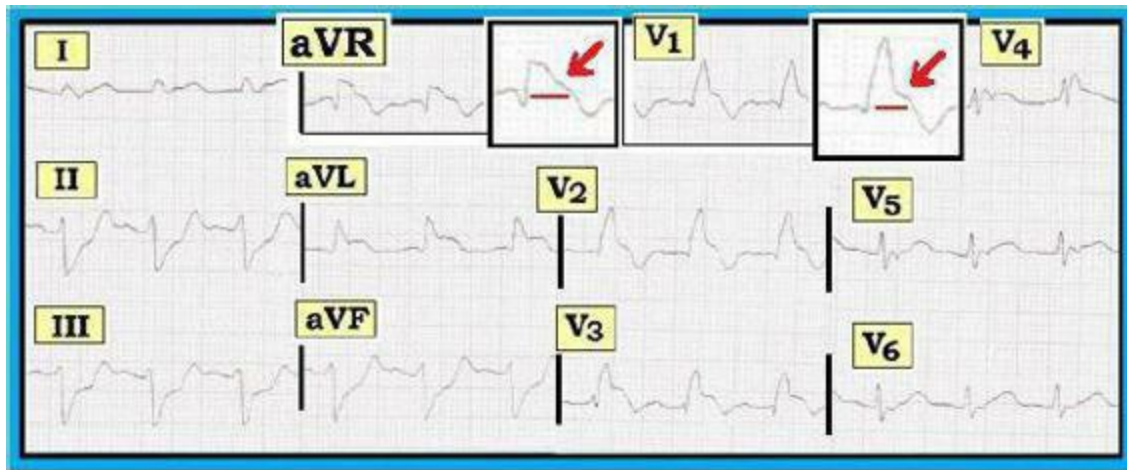


Figure 09.40-2: ECG from an acutely ill patient with *new-onset* chest pain. New *bifascicular* block in context with *dramatic* ST-T wave changes seen here (including **ST elevation** in lead aVR and in lead V1 — red arrows) — suggest acute *proximal* occlusion of either the LAD or LCMA (See text).

We conclude Section 09.40 with the ECG shown in **Figure 09.40-3** — obtained from a woman with chest pain and heart failure. We previously encountered this ECG in Section 09.28 — at which time we focused attention on the *diffuse* ST segment depression that is seen (red arrows).

- **NOTE:** In addition to *diffuse* ST depression — there is also *significant* ST elevation in **lead aVR** of Figure 09.40-3. Is this patient likely to have severe CAD because there is diffuse ST depression with ST elevation in lead aVR?

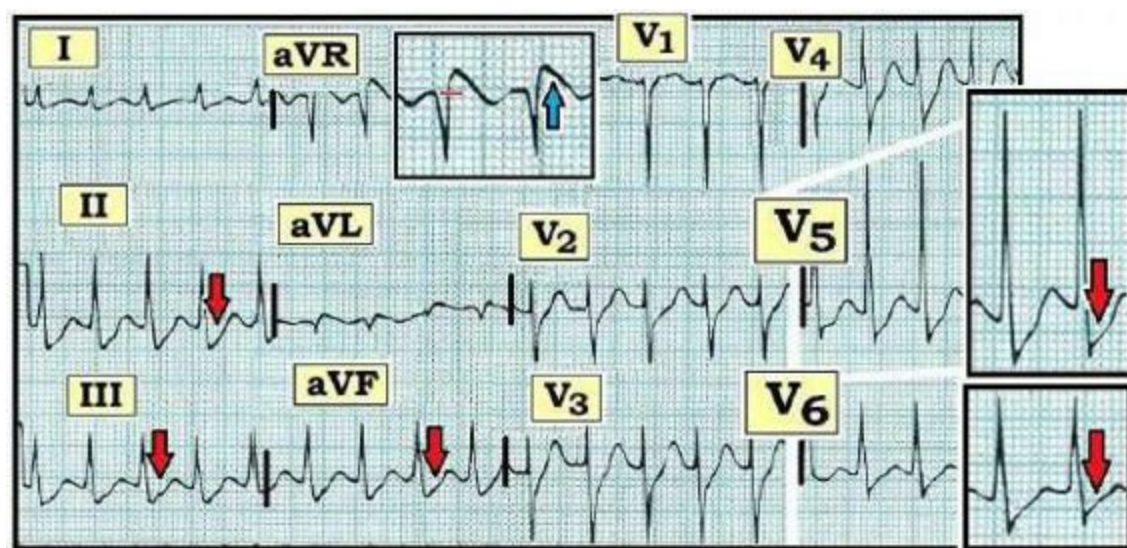


Figure 09.40-3: ECG obtained from a woman with chest pain and heart failure (reproduced from Figure 09.28-1). Note *diffuse* ST depression with ST elevation in lead aVR. Is this patient likely to have severe CAD? (See text).

Answer to Figure 09.40-3: As discussed in Section 09.28 — the rhythm is PSVT at a rate of ~180/minute. Although *diffuse* ST depression is present in association with ST elevation in lead aVR — this does not necessarily indicate significant coronary artery disease because there is *marked* tachycardia. Instead — each of the entities in **LIST #4** (*Section 09.26*) should be considered as possibly contributing to the ST depression that is seen. The list of common causes of ST depression includes: **i)** Ischemia; **ii)** “Strain”; **iii)** Digitalis effect; **iv)** Low serum K⁺/Mg⁺⁺; **v)** Rate-related changes (*from tachycardia*); and/or **vi)** *Any combination* of causes i) *through* v).

- We advise *against* even contemplating the possibility of severe CAD until after resolution of the tachycardia. Then *repeat* the ECG.

10.0 – Acute MI / Ischemia



Acute MI (and *Ischemia*)

10.1 – The Patient with Chest Pain: *WHY Do an ECG?*

One of the *KEY* reasons for obtaining an ECG is to help *evaluate* the patient with *new-onset CP* (*Chest Pain*). By doing so we hope to determine: **i) IF** there are *any acute ECG changes?* and **ii)** Is there evidence of *prior MI* (*Myocardial Infarction*)? Specifically — We want to know:

- Is there *ongoing ACS* (*Acute Coronary Syndrome*)? This is determined by *history* — *troponins* — and by assessment of *acute and serial ECG changes*.
- If there is ACS — *What* area(s) of the heart are involved? — How *extensive* is the area of involvement?
- What is the cardiac rhythm? (*Any arrhythmias?*).
- Are there conduction defects? (*BBB? AV block?*).
- KEY: Is patient a *candidate* for *acute intervention*?

10.2 – *What is a “Silent” MI?*

As many as **1/3** of *all* infarcts are *“silent” MIs* — which means that these infarcts are *not* associated with chest pain. Approximately *half* of this group (ie, *~1/6 of all MI patients*) — have *“other”* symptoms (*but not chest pain*). These *non-chest-pain-equivalent symptoms* may include: **i)** acute dyspnea (*shortness of breath*); **ii)** GI symptoms; **iii)** vague myalgias or *flu-like* syndrome; or **iv)** mental status change (*especially confusion*). The *other half* of this group (*~1/6 of all MI patients*) — have *no symptoms* at all.

- Although *“silent” MI* is more common in the elderly — this entity may occur in *any* age group (*regardless of whether or not the patient has diabetes or other medical issue that might impair sensation*).
- By far — the most common of the *non-chest-pain equivalent* symptoms is shortness of breath! For this reason — We advise obtaining an **ECG** on virtually *any* adult of a certain age who presents with **unexplained new-onset dyspnea**. A “silent” MI may have precipitated pulmonary symptoms/heart failure.
- Awareness of the surprising *prevalence* of “silent” MI in the general population — should prompt you to inquire about a *possible “event”* (ie, *infarction*) whenever the patient presents with *unexplained* symptoms that might be attributable to an *undetected* recent infarction. Examples include: **i)** Recent *new* edema/heart failure but without a history of chest pain; and **ii)** *New* fatigue and/or confusion in an *older* patient without plausible explanation. Have a **low threshold** to obtain an **ECG** on such patients — even though they are *not* having chest pain.
- **Comparison** with a *prior ECG* may be *invaluable* for determining **IF** abnormalities seen on a current ECG are new or old. **IF** *old* records are *not* readily available — Use fax or cell phone

transmission to *expedite* the process.

- **Bottom Line:** The entity of “*silent*” MI is much more *common* than is generally appreciated. Not all “*silent*” MIs are truly “*silent*”. Instead — **other symptoms** (*especially shortness of breath — but also confusion, malaise, etc.*) are commonly associated with this entity. Maintaining a **high index of suspicion** is essential in order not to overlook *previous* MIs that may have been subtle and without the usual symptom of *cardiac-sounding* chest pain. Comparison of the patient’s *current* ECG with a **prior** ECG may be invaluable and indicate that a patient you thought had no prior history of heart disease has actually *had* an MI at some time in the past.

10.3 – The ECG in Acute MI: What are the Changes?

Recent years have seen *increased* emphasis on use of the ECG for evaluation of ACS. We look for **acute ECG changes** (Figure 10.3-1). The goal is to determine ASAP from *wherever* the patient is first seen — as to whether **acute intervention** is likely to be beneficial:

- The **KEY** is to recognize **acute STEMI** (**ST Elevation Myocardial Infarction**) at the earliest opportunity — because this is the group of patients most likely to benefit from **acute reperfusion** (*angioplasty/stenting*) of the occluded vessel.
- **Once acute STEMI is identified** — the process of *notifying* cardiology and/or *activating* the cath lab is set into motion. This will *ideally* be accomplished within *minutes* of obtaining and interpreting the patient’s *initial* ECG.
- Most patients with **NSTEMI** (**Non-ST Elevation MI**) — do not need acute intervention. The reason for *expediting* assessment of the *initial* ECG is to find those who do.
- Realize that what *begins* as a *non-ST-elevation* picture — might *evolve* into an *acute STEMI*. This is the reason for having a *low* threshold for **repeating the ECG** (*sometimes more than once*) when evaluating a patient with new-onset *ongoing* chest pain. **NOTE:** Acute ECG changes may evolve in as little as 20-30 minutes.
- **ECG Indicators of Acute MI** — are shown in **Figure 10.3-1**:

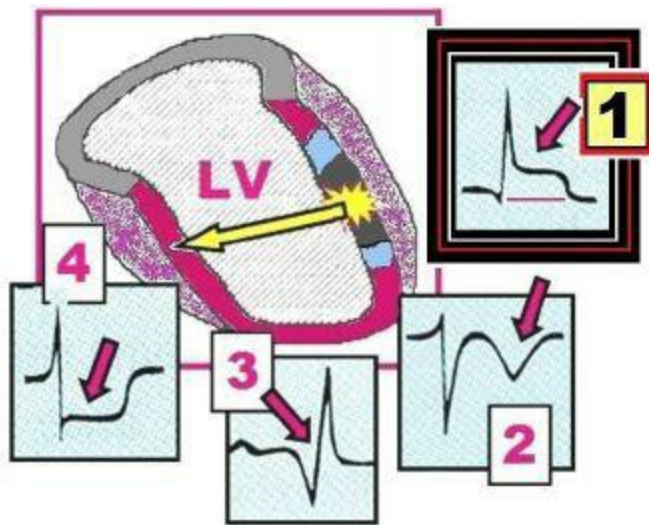


Figure 10.3-1: ECG Indicators of *Acute* MI: 1) ST segment elevation; 2) T wave inversion; 3) Q wave development; and 4) *Reciprocal* ST depression in *other* lead areas.

10.4 – ECG Indicators: 1) *ST Segment Elevation*

The hallmark of acute coronary artery occlusion — is **ST segment elevation**. This ST elevation is typically seen in the leads that *overly* the area of acute infarction (**Panel 1** in Figure 10.3-1). In general — the *greater* the amount of ST elevation and the *more* leads showing ST elevation — the *larger* the size of the acute MI (*and the more the potential for benefit from acute reperfusion*).

- CAVEAT #1: *Not* all ST elevation is the result of *acute* MI. **Other Reasons** for **ST elevation** that may *not* be due to *acute* STEMI include: i) *Early* repolarization (*Section 09.19*) ; ii) *Ventricular* aneurysm (*suspected* when ST elevation persists for months *after* a previous large infarction); iii) *Acute* pericarditis (*Section 12.0*); and iv) **Other conditions** (*patients with LVH; LBBB; IVCD; cardiomyopathy may at times manifest long-term ST elevation in certain leads not due to acute stemi*).
- CAVEAT #2: **STEMI “equivalent” patterns** exist. The most notable example of this — is *acute posterior* MI that manifests *anterior* ST depression *instead* of elevation (*Section 10.33*); *Clinical* implications of a “*stemi-equivalent*” — are *identical* to those of *frank* STEMI.

10.5 – ECG Indicators of *Acute* MI: 2) *T Wave Inversion*

Coronary *ischemia* is suggested by the ECG finding of **symmetric T wave inversion** (*as seen in Panel 2* in Figure 10.5-1). In general, the *deeper* the T wave inversion and the *more* leads involved — the *more extensive* the area of involvement is likely to be.

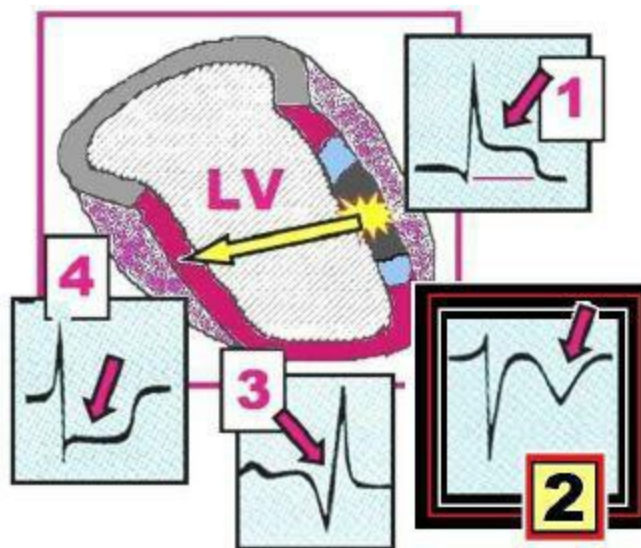


Figure 10.5-1: ECG Indicators of *Acute* MI (*reproduced from Figure 10.3-1*). In the paragraphs below, we highlight assessment of *symmetric* T wave inversion (**Panel 2**).

While emphasizing *pattern* recognition of **symmetric T wave inversion** as seen in **Panel 2** of Figure 10.5-1 — it is well to recall several caveats:

- CAVEAT #1: In addition to ischemia — there are **other reasons** for **T wave inversion**. These

include: **i)** LVH or RVH with “strain” (Section 08.9 and Section 08.28); **ii)** Acute pulmonary embolus (Section 08.37); **iii)** Juvenile T wave variant (Section 08.31); **iv)** **Other conditions** (medications; electrolyte disorders; BBB; cardiomyopathy; longstanding coronary artery disease; and various non-cardiac conditions may all manifest shallow or deep T wave inversion not due to an acute ischemic process); plus **v)** A **normal variant pattern** (Isolated symmetric T inversion may sometimes be seen as a normal variant in leads III, aVF, aVL, aVR, and/or VI — Section 09.12).

- **CAVEAT #2:** One can not tell IF even deep symmetric T inversion is new or old from looking at a single ECG. **Comparison** with a **prior tracing** is needed. The interpreter simply *describes* what is seen.
- **CAVEAT #3:** Even when *symmetric* T wave inversion is indicative of *acute* ischemia in a patient with *new-onset* chest pain — this does not necessarily mean that an *acute* MI is evolving. A *tincture* of time — **serial ECGs** — **troponins** and **clinical follow-up** are all needed to determine IF symmetric T inversion present on *initial* ECG will *evolve* into a *Q-wave* or *non-Q-wave* infarction.

Consider the two 6-lead sequences shown in **Figure 10.5-2**. In *both Panel A and Panel B* — there is **symmetric T wave inversion** in lead III.

- Which of these 2 sets of limb leads is more likely to represent ischemia?

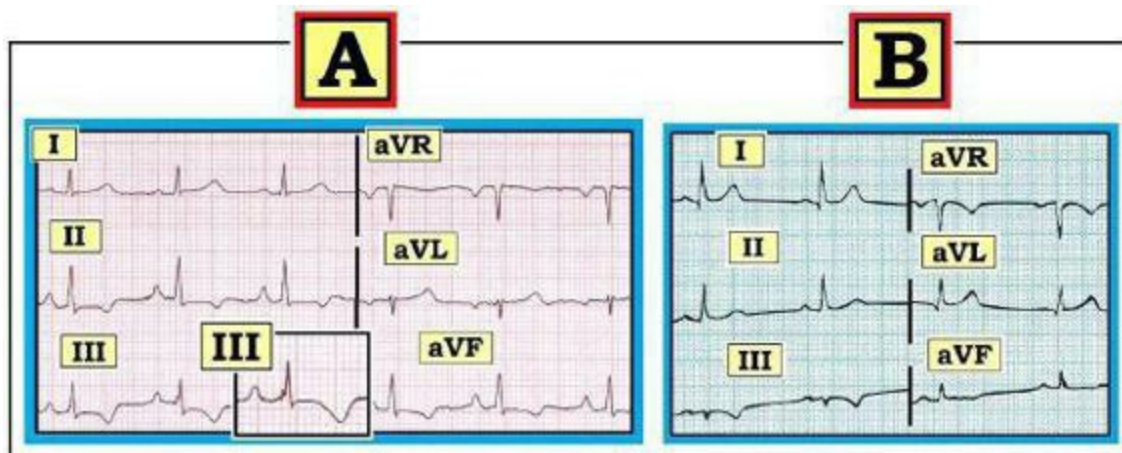


Figure 10.5-2: Ischemic T wave inversion. **Panel A** — shows fairly deep, *symmetric* T wave inversion in lead III. T inversion in the *other* inferior leads (*leads II and aVF*) is not quite as deep — but it is present. We therefore interpret the picture in **Panel A** as highly suggestive of **inferior ischemia**. In contrast — **Panel B** (which we have reproduced from Figure 09.13-1) is much **less suggestive of ischemia** because: **i)** T inversion is minimal (*if present at all*) in lead aVF and absent in lead II; plus **ii)** the QRS complex is predominantly *negative* in lead III. This could be normal — since the T wave vector often follows close behind the QRS vector (Section 09.13). Clinical correlation will be important to further assess these assumptions (See text).

Answer to Figure 10.5-2: The picture of ST segment *coving* and *symmetric* T wave inversion in lead III is of potential concern for *both* Panel A and Panel B. That said — we are clearly **more concerned**

with **Panel A**.

- ST elevation or depression is always of more concern when **similar findings** are found in **neighboring leads**. Whereas T inversion/ST depression is seen in *each* of the *inferior* leads in **Panel A** (II,III,aVF) — T inversion is *isolated* to lead III in **Panel B** (*with no more than nonspecific ST flattening in lead aVF*).
- **Lead III** — is one of the leads that may *normally* manifest even *moderate-to-large* T wave inversion, without this being due to ischemia (*Section 09.12*). That this is the case in **Panel B** — is made more likely by the associated finding of a **predominantly negative QRS** complex in the lead showing T wave inversion.
- **NOTE:** We can *not* exclude the possibility of acute ischemia (*or even ongoing infarction*) from the limb lead sequence shown in **Panel B**. Clinical correlation and comparison with prior ECGs is needed to do so. But our suspicion of ischemia/infarction is clearly *less* for Panel B than it is for the limb lead sequence in **Panel A**.

10.6 – ECG Indicators of Acute MI: 3) Q Waves

Development of **Q waves** has long been thought of as the **marker** that **myocardial infarction** *has* taken place (**Panel 3** in *Figure 10.6-1*). In general, the *deeper* and *wider* a Q wave is and the *more* leads in a given lead area that manifest Q waves — the *more* likely it is that this ECG sign indicates ongoing or prior infarction. That said — there are important caveats to be aware of:

- **CAVEAT #1:** *Not* all patients with *acute* MI develop Q waves. Instead, there are **non-Q-wave infarctions** — in which *acute* MI is documented by history, *evolutionary* ECG changes and *positive* cardiac markers (**troponins**).
- Among patients who *do* develop Q waves with infarction — transmural (*full-thickness*) involvement is *not* necessarily needed for a Q wave to form.
- **CAVEAT #2:** When Q waves *do* develop — they do *not* necessarily last. Instead — Q waves may decrease in size with time. *They may even disappear*. As a result — the term, “**significant Q wave**” is **problematic**. Even a *small* Q wave may be “*significant*” in that it could be the *only* ECG sign remaining from a *prior* infarction.
- **CAVEAT #3:** There are **other reasons** for **Q waves** apart from serving as a marker of infarction. These include: **i)** Scarring, fibrosis, cardiomyopathy; **ii)** Lead placement errors (*Section 09.8*); **iii)** **Other conditions** (*patients with LVH; RVH/COPD; LBBB; LAHB; IVCD; chest wall deformity may all manifest poor R wave progression with resultant precordial Q waves — as listed in Figure 09.7-1*); plus **iv)** **Normal Variant** (*Be this in the form of normal small septal q waves or even moderate-to-large isolated Q waves in leads III, aVF, aVL, aVR, and/or V1*). Recognizing when Q waves are likely to reflect a *normal* finding was discussed in *Section 09.12*.

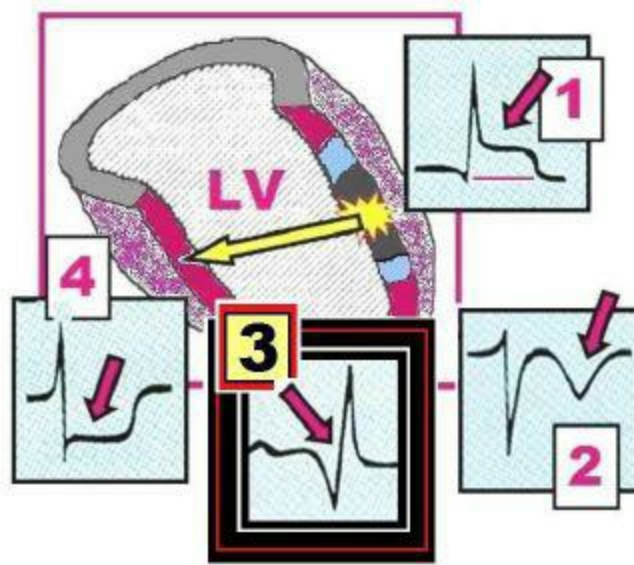


Figure 10.6-1: ECG Indicators of *Acute MI* (reproduced from [Figure 10.3-1](#)). In the paragraphs below, we highlight development of Q waves (**Panel 3**).

10.7 – Q Waves: *Why Do they Form?*

The theory for genesis of Q waves is simple (**Figure 10.7-1**):

- After depolarization of the ventricular septum — **activation of *ventricular myocardium*** begins. If one thinks of the LV (*Left Ventricle*) as a *cylindrical* structure — the *initial milliseconds* of the activation process are directed *outward* everywhere as the electrical impulse begins on its path from *inner* endocardium (*Endo*) — to the *outer* epicardial (*Epi*) layer of the heart (**Panel A** in [Figure 10.7-1](#)).

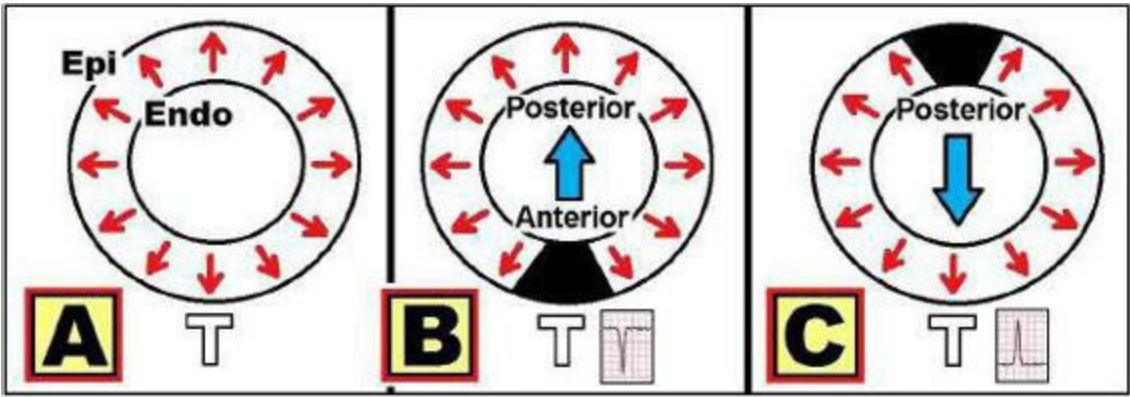


Figure 10.7-1: Genesis of Q waves. *Schematic* cross-sectional view of the heart. *Initial* electrical activity normally *cancels out* — as electrical activity is *opposing* everywhere (**Panel A**). The reason a **Q wave** forms over the area of infarction is that *null* electrical activity from myocardial necrosis is *no longer* opposed (**Panel B**). With **posterior infarction** (shown in **Panel C**) — a **tall R wave** is seen in *anterior* leads because of *loss* of posterior forces (See text).

Looking Closer at Figure 10.7-1: An important concept to emphasize — is that the overwhelming majority of electrical forces involved in activation of the heart cancel each other out. The ECG waveforms we see on an electrocardiogram represent the *less than 10%* of electrical forces are *not* directly opposing each other. This concept is well illustrated in **Panel A** of *schematic* [Figure 10.7-1](#)

— in which *virtually all* electrical forces during the *initial* milliseconds of ventricular depolarization cancel each other out (*Note arrows in Panel A oppose each other in all directions — resulting in a null vector for the very first instant during ventricular activation*).

- **Panel B** — schematically depicts what occurs during the initial milliseconds of ventricular activation in the presence of **anterior infarction** (*black area in Panel B*). The reason a **Q wave** develops over **anterior leads** — is that *outward-directed* posterior forces are now *unopposed* because of the null vector over the anterior area of infarction.
- **Panel C** — depicts what occurs with **posterior infarction** (*to be discussed in detail in Sections 10.33-through-10.37*). In this case — the *null* electrical vector (*black area*) lies posteriorly over the area of infarction. The result is *unopposed* forces that move anteriorly. This generates a **tall R wave** in **anterior leads**.

10.8 – ECG Terminology: *Distinction between Q, q and QS waves?*

You will often be asked to *verbally* describe the ECG findings noted on a given tracing. This becomes especially relevant when evaluating patients for acute or prior infarction. It is therefore important to clarify *semantics* of the **ECG Terminology** that is most commonly used for description (**Figure 10.8-1**):

- If a *negative* deflection *precedes* the QRS complex — it is termed a **Q wave**.
- The first *upward* deflection of the QRS complex is termed an **R wave**. If this is followed by a *second* upward deflection (*as occurs in lead VI with RBBB*) — it is called an **R'** (*R prime*).
- The downward deflection that *follows* the R wave is termed an **S wave**. (**NOTE:** *An S wave is only said to be present — IF the negative deflection after the R wave descends below the baseline*).
- **KEY Point:** Given the importance of **Q waves** as a “*marker*” of **infarction** — the *initial* direction of the QRS complex should be *closely* scrutinized to determine **IF** it is up (*an r wave*) or down (*a q wave*).

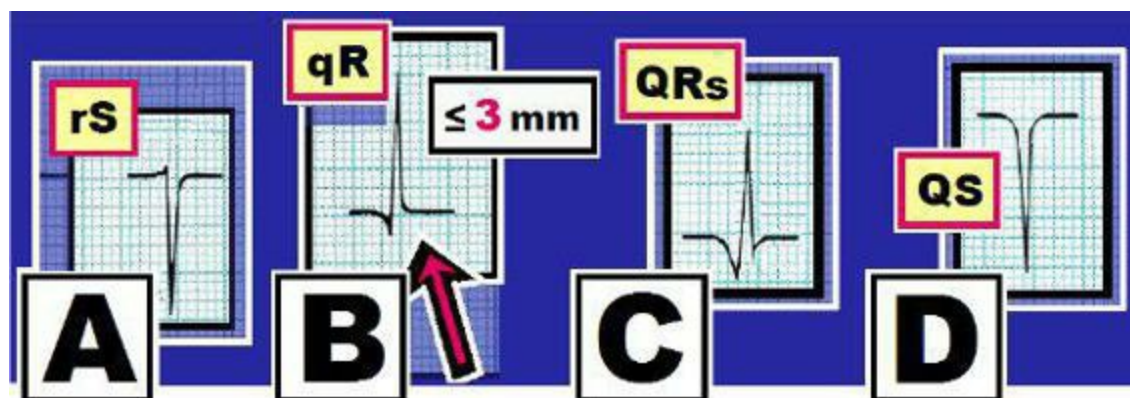


Figure 10.8-1: QRS Nomenclature. Although tiny — the *initial* QRS deflection in **Panel A** is upward. This deflection is therefore an **r wave**. In **Panel B** — the *initial* QRS deflection is *downward*. It is therefore a **q wave**. A **lower case** designation is used since this q wave in Panel B is *less than 3mm* in depth. In **Panel C** — there is a **QRs** complex (*a wide and deep Q wave but a small*

and narrow terminal s wave). Since there is *no* R wave at all in **Panel D** — we describe the complex seen as a **QS** complex (*See text*).

Large vs ***Small-Case Designation*** — *Large* deflections are denoted by *capital* letters. In contrast — ***smaller deflections*** (*that do not exceed 3 mm = 3 little boxes*) are denoted by ***lower-case letters***. Thus, we designate the QRS complex in **Panel A** of **Figure 10.8-1** as an “**rS**” complex (*tiny initial r wave; deep S wave*). **NOTE:** The importance of recognizing that the *initial* deflection in **Panel A** is upright — is that *without* this tiny initial *positive* deflection, there would be a Q wave.

- **Panel B** — is a **qR** complex. The initial *negative* deflection is small and narrow (*a q wave*). This is followed by a tall upright (*R wave*) deflection.
- **Panel C** — is a **QRs** complex. The initial deflection is not only deep, but also quite wide. We call it a ***large Q wave***. The tiny *negative* deflection following the tall R wave in **Panel C** is a small terminal s wave.
- **Panel D** — is designated a “**QS complex**”. Because there is *no* positive deflection (*R wave*) in **Panel D** — we are *unable* to distinguish between a Q wave, an S wave, or some *combination* of the two. **QS complexes** are commonly seen in the ***anterior leads***. They may or may not serve as a marker of infarction (*Section 09.10*).

10.9 – Summary: *When are Q Waves Normal?*

We summarize below **KEY** points brought out in Sections 10.6, 10.7, 10.8 — regarding ECG assessment for the presence and significance of **Q waves**:

- The ***bigger and wider*** a **Q wave** is — the more likely it is that infarction has at *some* time occurred. This is especially true **IF** there is *more* than one Q wave in a given lead area. That said — *some* leads may *normally* manifest even *large* Q waves that are *not* necessarily indicative of infarction. These leads are III – aVR – aVL – aVF – and V1 (*See **Figure 10.9-1** — previously discussed in Section 09.12*).
- **NOTE:** Infarction Q waves may get smaller (*and even disappear*) with time. As a result — *small* size of a Q wave does *not* necessarily mean that no infarction has occurred.
- Small ***septal q waves*** — may *normally* be seen in one or more of the ***lateral leads*** (*leads I, aVL; V4, V5, V6*). These *septal q waves* should *not* be confused with infarction q waves.
- ***Clinical correlation and comparison*** with ***prior tracings*** is essential for optimal assessment of the meaning of Q waves. For example — Seeing small and *narrow* q waves limited to *lateral* leads in an otherwise *healthy* patient *without* acute ST-T wave changes strongly suggests that these q waves are benign.

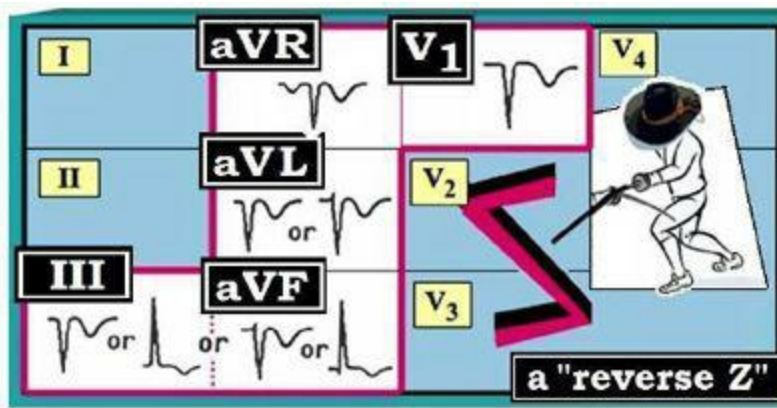


Figure 10.9-1: “Reverse Z” memory aid (reproduced from Figure 09.12-1) — for recalling the 5 leads that may display even *large Q waves and/or T inversion* as an *isolated* finding in otherwise healthy adults who do not have heart disease (See text and Section 09.12 for more details).

10.10 – ECG Indicators of Acute MI: 4) ST Segment Depression

We emphasized in Section 10.4, that with **acute coronary occlusion** — the ECG leads that *overly* the area of infarction manifest **acute ST elevation**. The opposite (*mirror-image*) picture is seen on ECG in *opposing* walls of the heart (**Figure 10.10-1**):

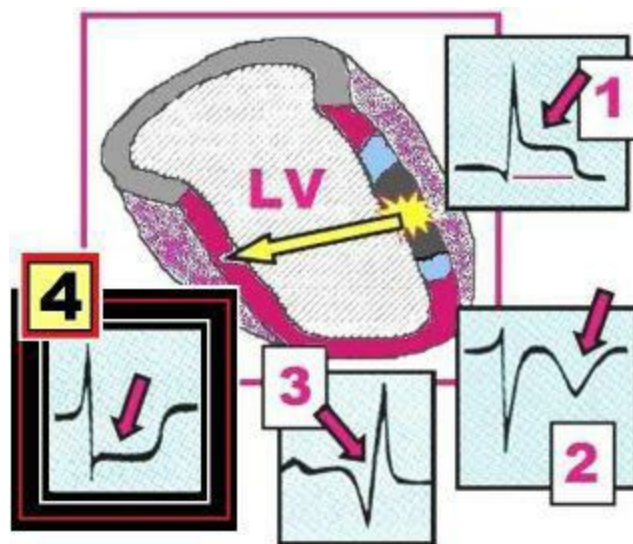


Figure 10.10-1: ECG Indicators of Acute MI (reproduced from Figure 10.3-1). With **acute STEMI** (**ST Elevation Myocardial Infarction**) — **ST elevation** is seen in leads that *overly* the area of acute infarction. The **mirror-image** picture (= **reciprocal ST depression**) is seen in *opposing* walls of the heart (**Panel 4**).

KEY Clinical Points: Given the *many* potential causes of ST elevation *other than* acute STEMI (Section 10.4) — the ECG finding of **reciprocal ST depression** often provides an **invaluable clue** that ECG changes are acute.

- *Reciprocal* ST depression is not found in either early repolarization or acute pericarditis. The *persistent* ST elevation of ventricular aneurysm is also rarely accompanied by more than minimal ST depression.
- The **strongest ECG evidence** that ST elevation is **truly acute** — is finding a virtual “**mirror-image**” picture of *reciprocal* ST depression. This concept is well illustrated in Figure 10.10-2 — in which one can readily appreciate how the *shape* of the *inferior* ST depression in **Panel A**

would take on the *shape* of the ST elevation in leads I,aVL — IF the tracing was to be *flipped over* (**Panel B** in Fig. 10.10-2).

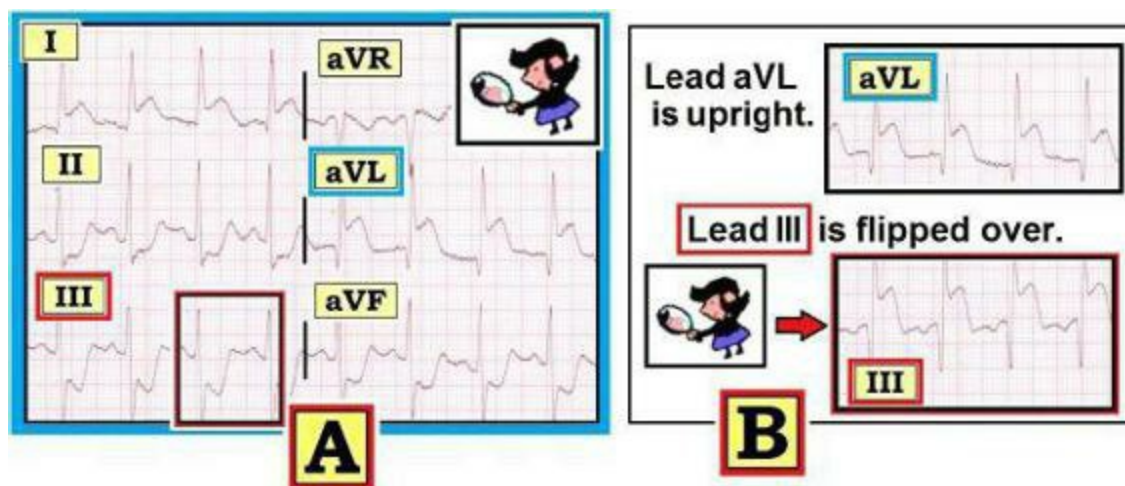


Figure 10.10-2: There is *marked* ST elevation in leads I and aVL. Note **mirror-image** (reciprocal) **ST depression** in *each* of the *inferior* leads (leads II,III,aVF). That is, the *depressed* ST-T wave seen in lead III from **Panel A** — would look virtually *identical* to the *elevated* ST-T wave shape in lead aVL IF in your mind’s eye you *flipped over* lead III (**Panel B**). With practice — you can readily recognize this **mirror-image** picture that is characteristic of *acute* reciprocal ST depression (See *text*).

Regarding Reciprocal ST Depression: The question arises as to *which* lead areas may manifest *reciprocal* ST depression with acute STEMI? Given the *cylindrical* configuration of the left ventricle — *any other wall* of the heart *not* showing ST elevation of acute STEMI **may manifest reciprocal ST depression**. That is, with acute *inferior* MI — *reciprocal* changes may be seen in anterior, lateral and/or posterior walls.

- As was the case for ST elevation (Section 10.4) — the *greater* the amount of *reciprocal* ST depression and the *more* leads showing ST depression — the *larger* the size the acute MI is likely to be (*and the more the potential for benefit from acute reperfusion*).
- **CAVEAT:** There are *many* potential **causes** of **ST depression** (Section 09.26). It will *not* always be easy to distinguish between ST depression due to **primary ischemia** or some *other* **cause** of ST depression (LV “strain”; drug effect; electrolyte disturbance) — vs **reciprocal changes** in a patient with *acute* infarction. That said — *clinical* correlation with assessment of the *overall* 12-lead ECG will usually indicate the clinical course to follow.

10.11 – Acute MI: The Sequence of ECG Changes



Textbooks describe a sequence of *evolutionary* changes that occur during the course of *acute* infarction. For those patients who “*read the textbook*” prior to having their *acute* MI — *schematic* **Figure 10.11-1** shows the typical *evolution* sequence to expect during the course of an *acute* STEMI (*ST Elevation Myocardial Infarction*):

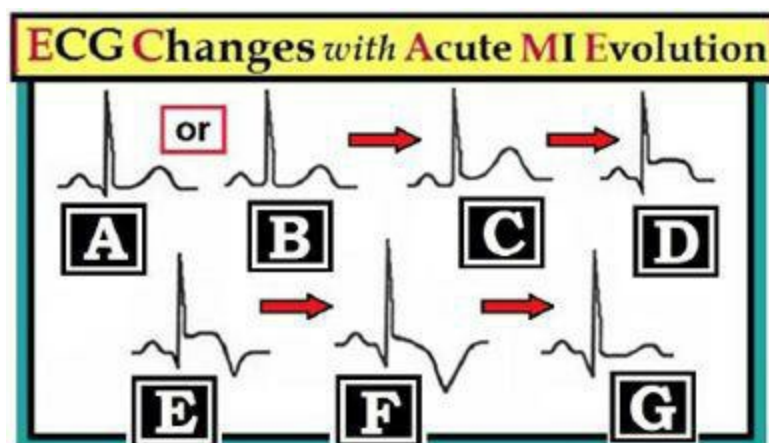


Figure 10.11-1: The typical *sequence* of ECG changes to expect during the course of *acute* STEMI (See text).

Regarding the *typical* sequence of ECG changes during *acute* MI:

- **Panels A and B** (in Figure 10.11-1) — are *both* normal complexes. We include Panel A to remind that small and narrow “*septal*” **q waves** may be a *normal finding* in one or more *lateral leads* (*I, aVL, V4, V5, V6* — Section 10.9). In contrast, the **Q waves** of *acute* MI tend to be bigger and wider — and infarction Q waves often *increase* in size during the course of *acute* MI.
- **Special NOTE:** On occasion — **small septal q waves** may also be seen in one or more *inferior leads* (*II, III, aVF*) as a *normal finding*. This is especially true if the frontal plane axis is relatively vertical (ie, *close to 90 degrees*).
- **Panel C** (in Fig. 10.11-1) — shows the “*hyperacute*” stage, which is the *earliest* change during *acute* MI evolution. This stage may be *subtle* — because there may be *no more than* minimal ST elevation. **Hyperacute T waves** — are recognized by being broader *and* peaked — almost as if the T wave is “*trying*” to *lift up* the ST segment. Awareness of *hyperacute* changes is important — because it allows recognition of *acute* STEMI-*in-progress* at a time when *acute* intervention/reperfusion may provide *optimal* benefit. The **hyperacute stage** is usually *short-lived* — and it may resolve *within* as little as 1-2 hours.
- **Panel D** — Conventional **ST elevation** follows (*with ST coving* = “*frowny*” shape) — and *developing* Q waves.
- **Panels E and F** — Q waves become bigger; ST segment elevation peaks and **T wave inversion** begins. T waves evolve as the ST segment returns to baseline (*shown in Panel F*). **NOTE:** As

opposed to the situation with **acute pericarditis** (Section 12) — the ST segment is often *still* a bit elevated with *acute* MI *at the time* that T wave inversion begins (**Panel F**). In contrast, with *acute* Pericarditis — the ST segment is *no longer* elevated in the *later* stage when there is T wave inversion.

- **Panel G** — ST-T wave abnormalities resolve (*or nearly resolve*) — but **Q waves persist** and serve as a **marker** that infarction has taken place.

10.12 – Variation in the Sequence of Acute MI Changes

The **A-thru-G** sequence from Figure 10.11-1 represents *expected* ECG findings for "**typical**" evolution of **acute** MI. That said — *Many patients do not read the textbook!* Variations on the general "theme" depicted in Figure 10.11-1 are common. Consider the following:

- **Q waves** do not always develop (Caveat #1 — *in Section 10.6*). There may be *non-Q-wave* infarction.
- When *infarction* Q waves *do* form — they may become smaller or even *disappear* over time.
- ST depression or T wave inversion *alone* may at times be the only ECG change that is seen. The appearance of these changes does not always follow the sequence shown in Figure 10.11-1.
- *Acute* MI may develop in an area of the heart that is **electrically "silent"**. For example — ECG changes may not always be seen (*or may be minimal*) when there is **high lateral** or **apical infarction**. *Special* leads may be needed to visualize **acute RV infarction** (Section 10.31).
- There may be **BBB** (Bundle Branch Block) or IVCD that was either present before or developed *as a result* of the acute MI. BBB may *mask* the ECG signs of acute or chronic infarction (Sections 05.24 through 05.29).
- There may be **acute posterior MI** — that produces a "**STEMI-equivalent**" pattern that manifests *anterior* ST depression rather than elevation (Section 10.33).

10.13 – KEY Points: ECG Changes of Acute MI

Despite the above cited potential variations in ECG presentation of acute *evolving* MI — awareness of the **typical** sequence shown in Figure 10.11-1 will go a long way toward optimally *rapid* recognition.

- Focus on **ASAP** recognition of **ST elevation** (*or stemi-equivalent*) patterns — since these are the patients who are *most* likely to benefit from **acute reperfusion** (*angioplasty/stenting*) of the occluded vessel. Even if positive troponins *later* confirm that infarction *did* occur — acute intervention is of limited (*if any*) benefit for *pain-controlled*, stable patients with non-STEMI (Section 10.3).
- Because ECG changes will not always be seen with *acute* MI and **troponins** may *initially* be *negative* — **History** becomes a **KEY** determinant for whether or not to admit a patient with chest pain to the hospital. As a general rule, IF at all in doubt — *Admit the patient*.
- Have a **low threshold** to **Repeat** the **ECG** — especially if the patient has *ongoing* chest pain or if you are *uncertain* about whether the *initial* ECG represents an *acute* event. An *acute evolving*

infarction may show changes in *as little* as 20-40 minutes!

- **Acute** ECG changes may be subtle (*as in the hyperacute stage* = **Panel C** in **Figure 10.11-1**). Look especially for **reciprocal ST depression** in lead areas not showing ST elevation to determine IF ECG findings are *likely* to be acute.
- Use the concept of "**patterns of leads**". That is, if *uncertain* about whether a Q wave or T wave inversion in **lead III** or **aVF** is *clinically* significant — look at the ***other inferior lead*** (*which is lead II*). ECG changes in the *inferior* leads are much *more* likely to reflect ischemia/infarction IF present in *each* lead of the grouping (ie, *in II, III and aVF*). Similarly — chest lead abnormalities are more likely to reflect ischemia/infarction IF present in *more* than a single lead.
- Use **Prior Tracings** to **compare**. *Go lead-by-lead*. Look not only at ST-T waves, but *also* at the QRS in *each* lead (*since axis change and/or lead placement errors may both affect ST-T wave appearance*).
- **NOTE:** A **change** in **QRS axis** from baseline to current tracing may alter ST-T wave appearance (*as well as resulting in Q wave appearance or disappearance*). Similarly — a **change** in the area of **transition** from baseline to current tracing may alter QRST appearance in precordial leads *without* this indicating that there has been interval change.
- **PEARL:** When the ECG was done — **Was the bed flat?** If not — Note the *angle* of the bed when the ECG was recorded. Many patients are too sick to lie flat (*Q waves and ST-T changes may be produced by even small amounts of bed incline, especially in the inferior leads*).

10.14 – Assessing *Acute* ECG Changes

Apply the concepts discussed in this section for assessing **acute ECG changes** to the tracing in **Figure 10.14-1** — obtained from a patient with *new-onset* chest pain.

- Is there evidence of *acute* infarction? If so — Is this an *acute* STEMI?
- Are there *reciprocal* changes?

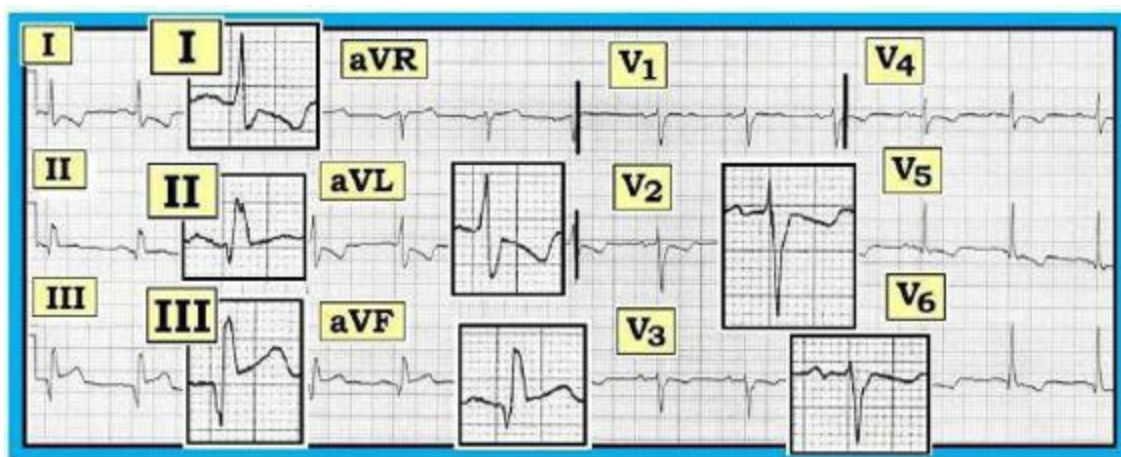


Figure 10.14-1: ECG from a patient with *new-onset* chest pain. Is there evidence of an *acute* STEMI? (See text).

Answer to Figure 10.14-1: The rhythm is sinus. All intervals and the axis are normal. No chamber

enlargement.

- **Q-R-S-T Changes:** There are large *inferior Q waves* (in leads II,III,aVF) — and small q waves in V5,V6. Transition occurs normally (*between V3-to-V4*) — but there is subtle *loss of R wave* from V2-to-V3.
- There is *hyperacute ST segment elevation* in leads III and aVF. By the concept of “*patterns of leads*” (ie, *simultaneously looking at all leads in a given lead group*) — there is probably *also* some ST elevation in lead II with *reciprocal changes* (ST depression/T wave inversion) in the *anterolateral* leads.

Clinical Impression: Sinus rhythm. **Acute inferior MI** (as diagnosed by the presence of Q waves; ST elevation; reciprocal ST depression). There is also probable **acute posterior MI** (positive “mirror test” in V2,V3 — See Section 10.33).

- The *above-noted* changes in Figure 10.14-1 qualify as an **acute inferior STEMI** — as there is ST elevation associated with *acute* ECG changes in this patient with *new-onset* chest pain. That said — it is difficult to “date” this infarction, because *inferior Q waves* are already well established (*they are especially wide and deep in lead III*). Whether this is the result of *prior* inferior MI with *superimposed* new acute MI — vs *recent* inferior MI with *acute* extension — vs *rapid* evolution of a single *ongoing* event is uncertain. **BOTTOM Line:** Clinical implications are similar *regardless* of whether the ECG in Figure 10.14-1 represents *old-plus-new* MI vs *single* new MI — since the *combination* of **new-onset chest pain plus acute ECG changes** (ST elevation; hyperacute T waves; reciprocal ST depression) means that the patient may benefit from *acute* reperfusion.
- The phenomenon of **Hyperacute T waves** is well illustrated in Figure 10.14-1 by T wave appearance in **lead III** (and to a lesser extent in lead aVF). Note that the T wave in these leads looks disproportionately tall and wide given the presence of ST elevation.
- **Reciprocal ST depression** — is also well seen in Figure 10.14-1 in *multiple* leads. In particular — Note the *mirror-image* appearance of the ST-T wave in lead III *compared to* lead aVL.
- **KEY Point:** The presence of both hyperacute T waves and reciprocal ST depression in *multiple* leads combine to strongly suggest that *despite* surprisingly deep and wide inferior Q waves — the ECG picture in Figure 10.14-1 is likely to be acute.

10.15 – FIGURE 10.15-1: Use of Serial ECGs in Acute STEMI

The ECG in Figure 10.15-1 was obtained from a patient with *new-onset* chest pain and **obvious acute STEMI**. Follow-up ECGs on this patient are shown in **Figure 10.15-2** (obtained a short while later) — and finally in **Figure 10.15-3** (obtained post-cath/reperfusion).

- Is there *evolution* of the MI on these *serial* ECGs?
- Was acute *reperfusion* successful (Figure 10.15-3)?

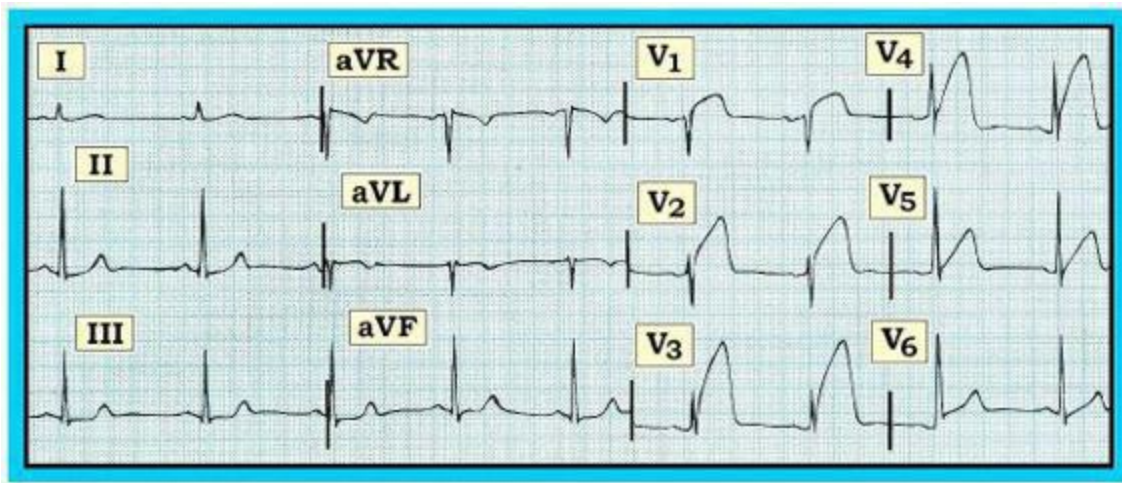


Figure 10.15-1: This is **ECG #1** (*blue border*) — from this patient with *new-onset* chest pain. There is sinus bradycardia with marked *precordial* ST elevation. Q waves have *not* yet formed in the *anterior* leads on this *initial* ECG #1. Note the *hyperacute* appearance of **ST-T waves** in leads V2,V3,V4. Surprisingly — *reciprocal* changes are minimal (*no more than slight ST-T wave flattening/depression in the inferior leads*). Despite this — there can be *little doubt* that this **ECG #1** represents a **large acute STEMI** in evolution.

What is the “Culprit” Artery?: We suspect acute *proximal* LAD occlusion as the “culprit” artery for the *acute* STEMI seen in Figure 10.15-1. As will be discussed in Section 10.25 — *proximal* LAD (*Left Anterior Descending*) occlusion is suggested by the ECG finding of *diffuse* precordial ST elevation that is especially *marked* in leads V2-to-V4.

- ***Beyond-the-Core:*** Another finding in favor of acute *proximal* LAD occlusion is that ST elevation is *more* marked in lead V1 than in lead aVR. In contrast — ST elevation tends to be more marked in aVR compared to V1 when there is left main disease.
- ***KEY Clinical Point:*** This patient is an *ideal* candidate for acute reperfusion — because there is *marked* ST elevation in Figure 10.15-1, but *no* anterior Q waves have yet formed. *Activate the cath lab ASAP!*

Two ***follow-up*** ECGs to Figure 10.15-1 are shown below. For clarity — We use a *different* color border for each tracing:

- Figure 10.15-1 — **ECG #1** (*blue border*) = the *initial* ECG obtained at presentation.
- Figure 10.15-2 — **ECG #2** (*red border*) = obtained a short while after ECG #1.
- Figure 10.15-3 — **ECG #3** (*green border*) = obtained after acute cath and angioplasty/stenting of the acutely occluded LAD.

As you evaluate these ***serial*** ECGs — Keep in mind the ***following*** Questions:

- Is there ECG evidence of *evolution* on these *serial* ECGs?
- Was acute *reperfusion* successful (Figure 10.15-3)?

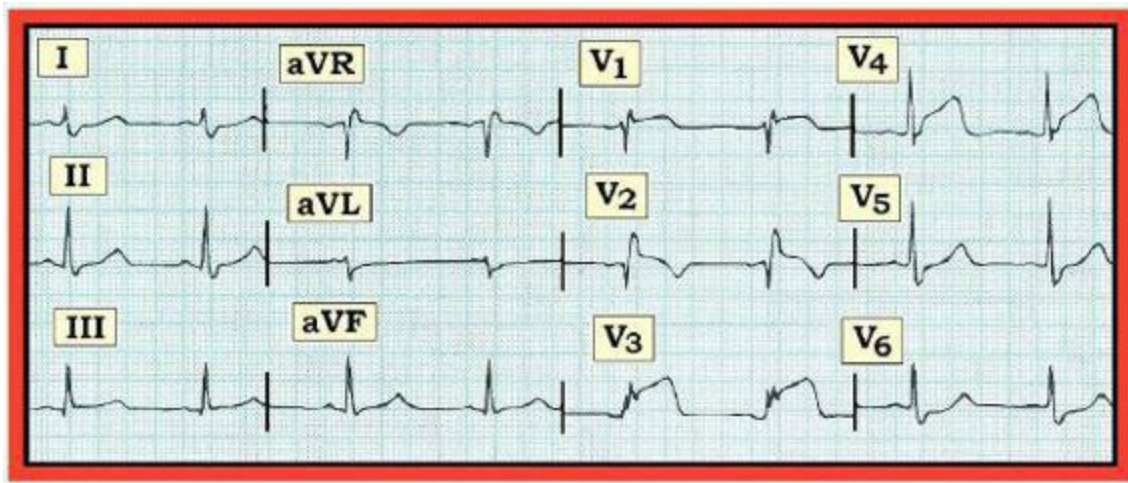


Figure 10.15-2: This is **ECG #2** (**red border**) — obtained a short while *after* ECG #1 from this patient with *acute* STEMI. Note that *since* ECG #1 — there has been interim development of **RBBB** (an *rSr'* complex is now seen in *V1* with wide terminal *S* waves in leads *I, V6*). The appearance of **lead V2** is concerning — as the large *new* **Q wave** and now **T wave inversion** in this lead suggest *ongoing evolution* is in progress (comparable to **Panel F** in **Figure 10.11-1**).

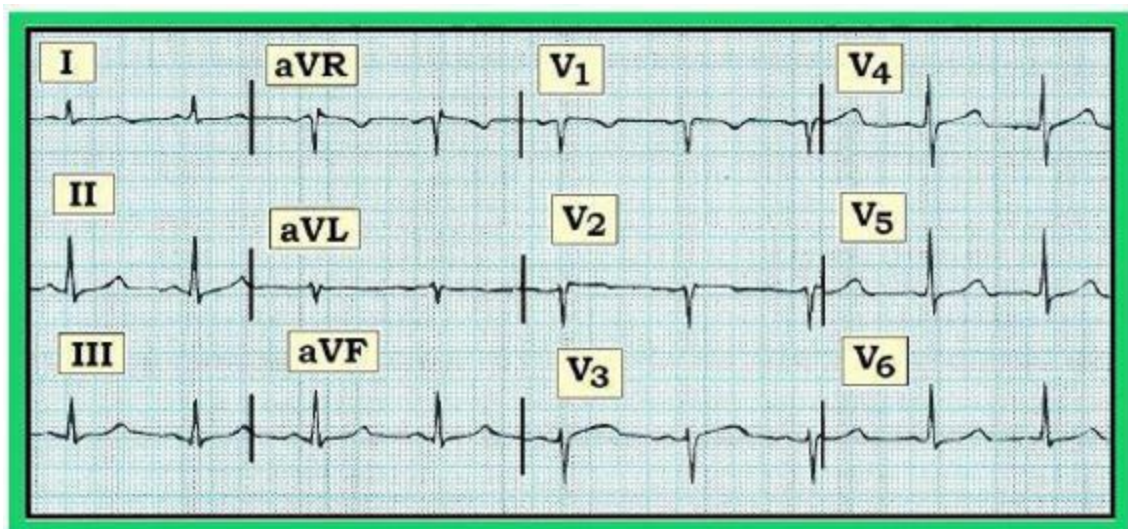


Figure 10.15-3: This is **ECG #3** (**green border**) — obtained after acute catheterization and angioplasty/stenting of the acutely occluded LAD. The “good news” — is that this *post-cath* **ECG #3** is encouraging! Note that the QRS complex has *narrowed* and RBBB is *no longer* present. The **Q wave** seen earlier in lead *V2* of **ECG #2** has *resolved* — and ST-T waves have essentially returned to baseline. R wave progression is essentially normal (*with transition between V3-to-V4*). It appears that *acute reperfusion* has **salvaged significant myocardium!**

BOTTOM Line: Use of *serial* ECGs may be extremely valuable in following the course of *acute* MI. *Lead-to-lead* comparison of QRS morphology and ST-T wave changes facilitates determining *which* changes are new — as well as providing insight to the likely benefit obtained from *acute* intervention.



The Coronary Circulation

The most common cause of *acute* MI is **sudden total occlusion** of a **major coronary artery**. The area of the heart affected will depend on distribution to the area from the coronary circulation. In these next few sections — We briefly review **normal coronary anatomy** with the goal of assisting in *rapid* identification of the “**culprit**” artery.

- Prompt recognition of *acute* coronary occlusion with *rapid* initiation of **reperfusion therapy** is essential for optimal outcome. For this — evaluation of the **initial ECG** is invaluable.
- Appreciation of normal coronary anatomy and common variants facilitates the process.
- **NOTE:** Basic core material review of the coronary circulation is presented in Section 10.17. For those wanting *additional* material — More advanced aspects of the coronary circulation that extends Beyond-the-Core — is covered in Sections 10.18 *through* Section 10.21.

10.17 – Overview of Normal Coronary Anatomy & Variants

In Figure 10.17-1 — We present a *schematic* overview of *normal* coronary anatomy (**Panel A**). Two of the most important anatomic variants are shown in Panels B and C:

- The two major vessels supplying the heart are the right and left coronary arteries. These most commonly arise from the right and left aortic sinuses, respectively (Panel A).
- In **most patients** (80-90%) — the **RCA** (Right Coronary Artery) is a **dominant vessel** that supplies the RV (Right Ventricle) — and then continues as the **PDA** (Posterior Descending Artery) along the *undersurface* of the heart (*unlabeled dotted vessel arising from the RCA in Panel A*) to supply the *inferior* and *posterior* walls of the LV (Left Ventricle). Not shown — the **AV nodal artery** is most often supplied from the RCA.
- As suggested in Panel A of **Figure 10.17-1** — the **LMain** (Left Main Coronary Artery) is typically a short vessel (10mm) that then **bifurcates** into the **LAD** (Left Anterior Descending Artery) and the **LCx** (Left Circumflex Coronary Artery).
- Normally — the **LAD** runs along the *anterior* epicardial surface of the heart in the interventricular groove on its path toward the cardiac apex. The **LAD** generally supplies: **i)** the *anterior* wall of the heart (*via its diagonal branches*); **ii)** the *cardiac* apex; and **iii)** a major portion of the conduction system (*via septal perforators that run vertically down through the septum*).
- The **LCx** (Circumflex) **Artery** — wraps around the *lateral* free wall of the LV (Left Ventricle). In most patients (ie, *when the RCA is dominant*) — the LCx becomes relatively *small* after giving rise to one or more **obtuse marginal branches** that supply the lateral free wall (*dotted vessels arising from the LCx in Panel A*).

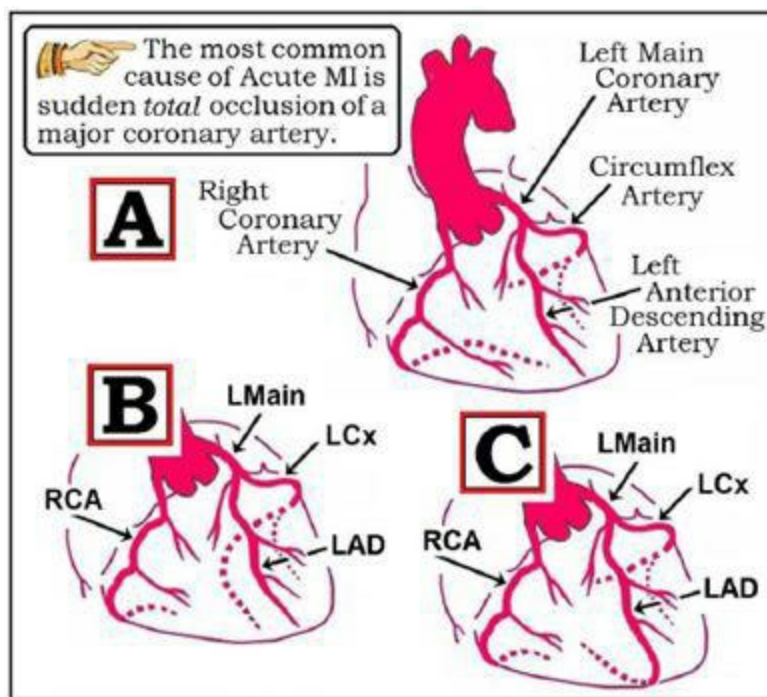


Figure 10.17-1: Overview of *normal* coronary anatomy. **Panel A** — the most common situation (80-90%), in which the **RCA** is a *dominant* vessel that supplies the RV as well as the *posterior* and *inferior* walls of the LV. The **LAD** normally supplies the *anterior* wall of the heart (via *diagonal branches*); part of the *cardiac apex*; and a major portion of the conduction system (via *septal perforators that run vertically down through the septum*). **Panel B** — represents a **left-dominant** circulation, in which the **LCx** (rather than the **RCA**) supplies the *posterior* and *inferior* walls of the left ventricle. **Panel C** — represents an **LAD “wrap-around”** variant, in which the LAD also supplies part of the *inferior* wall as well as the *anterior* wall (See text).

Common Variants in Normal Coronary Anatomy: Figure 10.17-1 is purely schematic. Realize that numerous variations exist on the most common picture of *normal* coronary anatomy that is shown in **Panel A**. These depend on *anatomic* differences and extent of disease (*development of collaterals*) — plus any revascularization that may have occurred.

- Anatomic appearance also depends greatly on **projection angle** for the **basic views** that are shown (ie, *septal perforator branches appear longer when viewed from a less superior angle*).
- Our discussion is limited to generalities.
- **Panel B** (in Figure 10.17-1) — represents a **“left-dominant”** circulation. This anatomic variant is seen in ~15% of patients. In such cases — the **RCA** is a **smaller** vessel than depicted in **Panel A**. To compensate — the **LCx** is typically **larger** and gives rise to the **PDA** (*large unlabeled dotted vessel arising from the LCx in Panel B*). In a **left-dominant** circulation — the **inferior** and/or **posterior wall** of the LV is supplied by the LCx.
- **Clinically** — the existence of a **left-dominant circulation** in ~15% of patients explains why *inferior* and *posterior* MIs will not always be due to acute RCA occlusion. Similarly, since the **AV nodal artery** may be supplied by the LCx in a *left-dominant* circulation — AV block may also occasionally occur with LCx (rather than RCA) occlusion.
- **Panel C** (in Figure 10.17-1) — represents a **“wrap-around”** **LAD** variant circulation. Occasionally — the **LAD** (**Left Anterior Descending Artery**) is **larger** and **longer**, to the point of extending beyond the cardiac apex and “wrapping around” to **supply** the **undersurface** of

the heart (*dotted vessel extending underneath the apex from the LAD in Panel C*). In extreme cases — a “wraparound” LAD may even serve the function of the PDA.

- Clinically — Awareness of the possibility of a “**wrap-around**” LAD circulation as an anatomic variant may explain the occasional ECG pattern of *simultaneous* ST elevation in ***inferior and anterior lead areas***. Not surprisingly — infarction the patient with a “wraparound” LAD variant may be quite large. Beyond-the-Core: Another reason for the occasional occurrence of *simultaneous* inferior and anterior ST elevation may be Takotsubo Cardiomyopathy (See Section 10.61).

10.18 – The RCA: Taking a Closer Look

In Figure 10.18-1 — We now take a *closer* look at normal (*expected*) coronary anatomy of the **RCA** (Right-Coronary Artery). We emphasize that this **closer look** at coronary anatomy constitutes ***advanced*** material for those wanting to know more. It is Beyond-the-Core for a basic ECG curriculum.

- As discussed in Section 10.17 — the **RCA** arises from the right anterior aortic sinus (**Figure 10.18-1**). The usual path of the RCA is along the right atrioventricular (AV) groove on its way to the “**crux**” (= *the point on the diaphragmatic surface where the anterior AV groove — posterior AV groove — and inferior interventricular groove all meet*).
- The 1st branch of the RCA is usually the **conus artery** which supplies the right ventricular outflow tract (*not shown on Figure 10.18-1*).
- The 2nd branch is usually the **SAN** (SA Nodal artery). Of note — the RCA supplies the SAN in ~60% of patients. In the remaining ~40% — the SAN arises either from the LCA or from *both* the RCA and LCA. Beyond-the-Core: Awareness of the fact that the SAN is most often supplied by the RCA — explains why **atrial infarction** is most often seen in the setting of acute *proximal* RCA occlusion (*in association with acute infero-postero and right ventricular stemi* — See Section 09.35).
- The **midportion** of the **RCA** gives off one or more **AM** (Acute Marginal) **branches** that supply the anterior wall of the RV (Right Ventricle). On occasion — AM branches may provide collateral circulation to the anterior wall when the LAD is occluded.

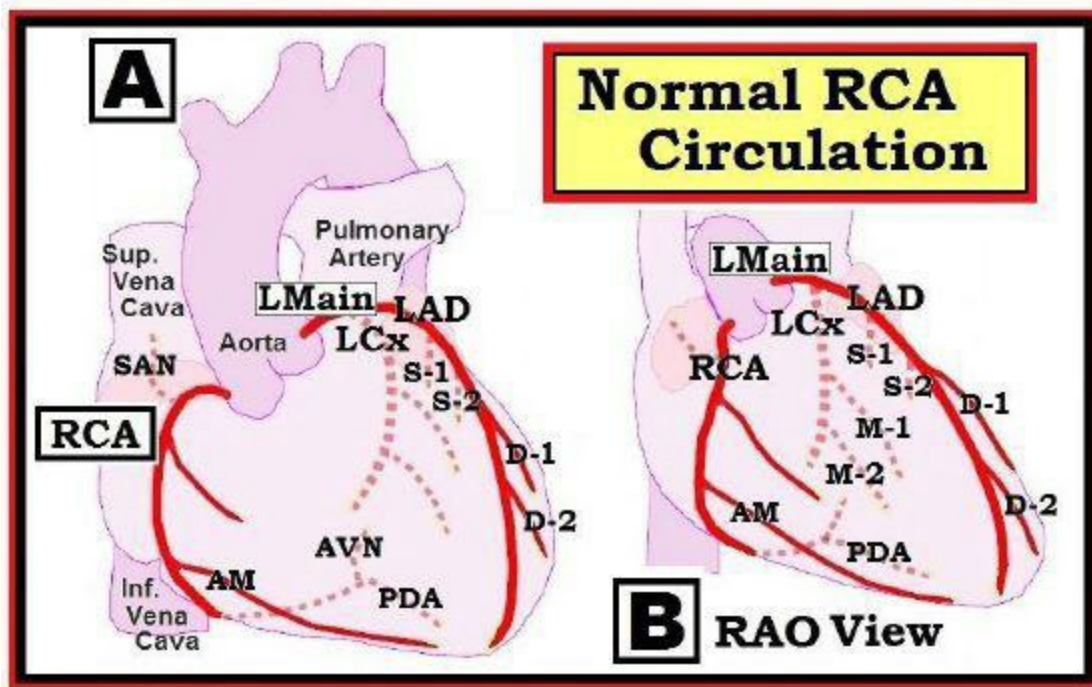


Figure 10.18-1: Normal coronary anatomy of the **RCA** (*Right Coronary Artery*) and its major branches. **Panel A** — anterior view. **Panel B** — RAO (*Right-Anterior Oblique*) view (See text). Abbreviations: **SAN** (*SA Nodal Artery*); **AM** (*Acute Marginal branches from the RCA*); **AVN** (*AV Nodal Artery*); **PDA** (*Posterior Descending Artery*).

Additional Notes on Figure 10.18-1: In *most* patients (80-90%) — the **RCA** is a **dominant vessel** that continues after giving off one or more **AM** branches as the **PDA** (*Posterior Descending Artery*) along the undersurface of the heart. The PDA supplies the **posterior** and **inferior** walls of the **LV** (*Left Ventricle*).

- The PDA also gives rise to a number of small inferior septal branches that travel *upward* to supply the lower septum, and which connect with septal branches traveling downward from the LAD (*not shown on Fig. 10.18-1*).
- On occasion — the **PDA** may also give rise to exceptionally large **PLA** (*Postero-Lateral Artery*) **branches** that extend laterally to supply parts of the LV free (*lateral*) wall. Beyond-the-Core: Awareness of the variant in which there may be large PLA branches arising from the PDA (*not shown on Fig. 10.18-1*) — explains why you may occasionally see acute *infero-postero-lateral* stemi (*with ST elevation in V5,V6*) from acute RCA *rather than* LCx occlusion (See Section 10.40.5).
- The **AVN** (*AV Nodal Artery*) is a branch of the RCA in about 2/3 of patients (**Panel A in Figure 10.18-1**). In the remainder of patients — the AVN arises from the LCA (*Left Coronary Artery*).
- Clinically — the reason AV Wenckebach (*Mobitz Type I 2nd-degree AV block*) is most commonly seen with acute *inferior* MI — is that that the AVN usually arises from the RCA.
- Beyond-the-Core: Mobitz I 2nd-degree AV block may occasionally be seen with acute LCx occlusion — in patients with a *left-dominant* circulation (*in which case the AVN is generally supplied by the dominant circumflex vessel*).
- Summary: **Acute occlusion** of the **RCA** — typically results in **acute inferior infarction**. Awareness of the most common anatomic RCA patterns provides insight to possible *associated* RV and *posterior* involvement — *as well as to potential SA nodal and AV nodal conduction*

abnormalities.

10.19 – The LEFT Coronary Artery: *Taking a Closer Look*

In Figure 10.19-1 — We take a closer look at normal (*expected*) coronary anatomy of the LCA (Left-Coronary Artery).

- As discussed in Section 10.17 — the LCA arises from the left aortic sinus (**Figure 10.19-1**). This vessel begins as the **LMain** (Left Main Coronary Artery), which is typically a short vessel (10mm) that then *bifurcates* into the LAD (Left Anterior Descending Artery) and the LCx (Left Circumflex Coronary Artery).
- Clinically — It is important to emphasize that **acute occlusion** of the LMain usually results in **rapid demise** of the patient (*since this generally leads to infarction of the entire left ventricle*). These patients typically die *before* reaching the hospital (See Section 09.40).

Major Branches of the LAD: The LAD (Left-Anterior-Descending) **Artery** runs along the anterior epicardial surface of the heart in the interventricular groove on its path toward the cardiac apex. The LAD generally supplies the *anterior* wall of the heart, the *cardiac* apex and a major portion of the conduction system.

- The **major branches** of the LAD are i) the Septal perforator vessels; and ii) Diagonal branches.
- **Septal branch anatomy** is highly variable. We show 2 septal branches in Figure 10.19-1 (**S-1; S-2**) — but instead there may be only one septal branch or many septal branches, depending on individual anatomy. The **1st septal branch** is typically the largest; its takeoff is generally *just after* the takeoff of the 1st diagonal branch.
- The **interventricular septum** is the most densely vascularized area of the heart. This is as it should be given the integral role of the septum in providing blood supply to the heart's conduction system. **Septal perforators** normally run a vertical path *downward* following their takeoff from the proximal LAD.
- *Downward* penetrating septal branches from the LAD typically connect with *upward* penetrating septal branches from the PDA branch of the RCA. In this way — there is usually a **network** of **collaterals** from both LCA and RCA systems in the event of disease in one system. How adequately collaterals from one system compensate for disease in the other is subject to individual variation (*as well as to how rapidly occlusive disease develops*).
- Clinical Note: Very **proximal LAD lesions** have been known as “**widow-makers**”. Especially if proximal to the 1st septal perforator (*and the 1st diagonal branch*) — these lesions are virtual “**left-main-equivalents**” because of the extent of injury and conduction system damage they cause.
- **Diagonal branch anatomy** is also highly variable. We show 2 diagonal branches in Figure 10.19-1 (**D-1; D-2**) — but there may be 1, 2, or 3 diagonal branches supplying the *anterolateral* wall of the heart. Occasionally — there is *no* diagonal branch per se, but rather a discrete **ramus intermedius** arising from *between* the LAD and LCx to supply the anterolateral surface

(not shown on Fig. 10.19-1). Typically — it is the **1st diagonal branch** that is the largest.

- Clinical Note Considerable variation in number and course of diagonal branch anatomy (*and the angulated path that these vessels follow*) may require multiple views on cath to determine if occlusion is present.

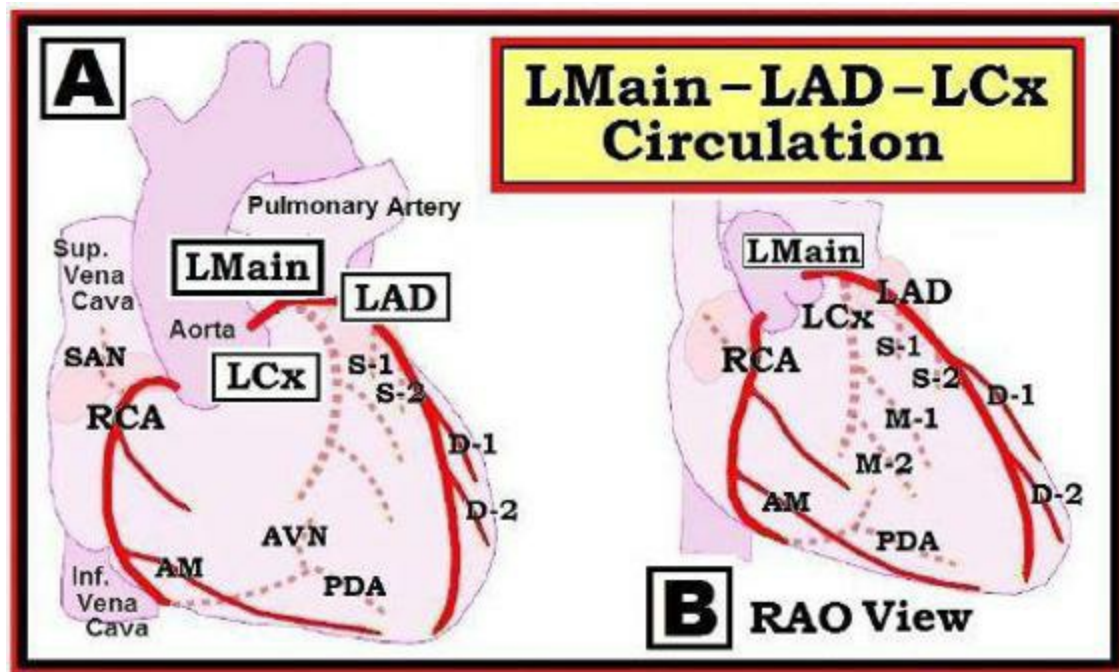


Figure 10.19-1: Normal coronary anatomy of the *left* coronary artery and its major branches. The LCA (*Left Coronary Artery*) begins as a short **LMain** (*Left Main Coronary Artery*) branch — which then *bifurcates* into the **LAD** (*Left Anterior Descending Artery*) and the **LCx** (*Left Circumflex Artery*). **Panel A** — anterior view. **Panel B** — RAO (*Right-Anterior Oblique*) view (See text). Abbreviations: S-1,S-2 (*Septal Perforator branches*) ; D-1,D-2 (*Diagonal branches*) ; M-1,M-2 (*Obtuse Marginal branches from the LCx*).

Major Branches of the LCx: The **LCx** (*Left Circumflex*) **Artery** is the other main branch of the LCA (Figure 10.19-1). In most (80-90%) patients — the LCx is limited in its course and primarily supplies the **lateral free wall** of the LV.

- After arising from the LMain — the **LCx** wraps around the lateral wall of the heart, and then typically runs in the left *posterior* atrioventricular groove in its path to the *infero-postero-interventricular* groove.
- The **main branches** of the **LCx** are the **Obtuse Marginals**. We show 2 *marginal* branches in Figure 10.19-1 (**M-1**; **M-2**) — but the usual number may vary from 1-to-3. In most patients — the LCx becomes relatively small after giving rise to its marginal branches.
- The **marginals** supply the **lateral free wall** of the LV (Think “marginal” = “lateral” free wall).
- Clinical Note Despite assessment of the **lateral wall** by no less than **5** of the 12 leads (**I,aVL; V4,V5,V6**) — the **high-lateral wall** may *not* be well visualized. This part of the LV is typically supplied by the **1st Diagonal branch** of the **LAD** (*and not by the LCx*).
- Beyond-the-Core: The lateral leads that most consistently assess the part of the heart supplied

by the LCx are leads V5,V6. Therefore — **ST elevation in V5,V6** most often suggests **acute LCx occlusion**. Exceptions — Some patients may have either large PLA (Postero-Lateral Artery) branches that arise from the RCA or they may have an *unusual* pattern of collateral circulation. Otherwise — acute ST elevation in V5,V6 = acute LCx occlusion.

PEARL: **ST elevation in lead aVL** may provide an *invaluable* clue to the location of the acutely occluded coronary artery. According to a study by Birnbaum et al (*Am Heart J* 131:38, 1996):

- *Suspect* acute **LAD occlusion proximal** to the **1st Diagonal** if *in addition* to ST elevation in aVL — there is *also* ST elevation in leads V2-through-V5. This is the most common situation when there is ST elevation in lead aVL.
- *Suspect* **1st Diagonal branch occlusion** if *in addition* to ST elevation in aVL — there is ST elevation in lead V2 (*but not in V3,V4,V5*).
- *Suspect* occlusion of the **1st Obtuse Marginal branch** of the **LCx** if *in addition* to ST elevation in aVL — there is ST depression in lead V2.
- **NOTE**: Anterior ST elevation without ST elevation in aVL — suggests LAD occlusion after takeoff of the 1st Diagonal (= D-I in Figure 10.19-1).

10.20 – LEFT-Dominant Circulation: Taking a Closer Look

Approximately 15% of patients have a “**left-dominant**” circulation. In such cases — the RCA is a *smaller* vessel, terminating before it reaches the crux. To compensate — the **LCx** is **larger** and **gives rise** to the **PDA** (Figure 10.20-1).

- Clinically — Awareness of the *left-dominant* anatomic variant accounts for the occurrence of most *infero-postero-lateral* infarction patterns.
- Beyond-the-Core: As noted in Section 10.18-1 — patients with a *left-dominant* circulation and *acute* LCx occlusion may manifest Mobitz I 2nd-degree AV block (*because the AV Nodal artery is usually supplied by the LCx when this vessel is dominant*).

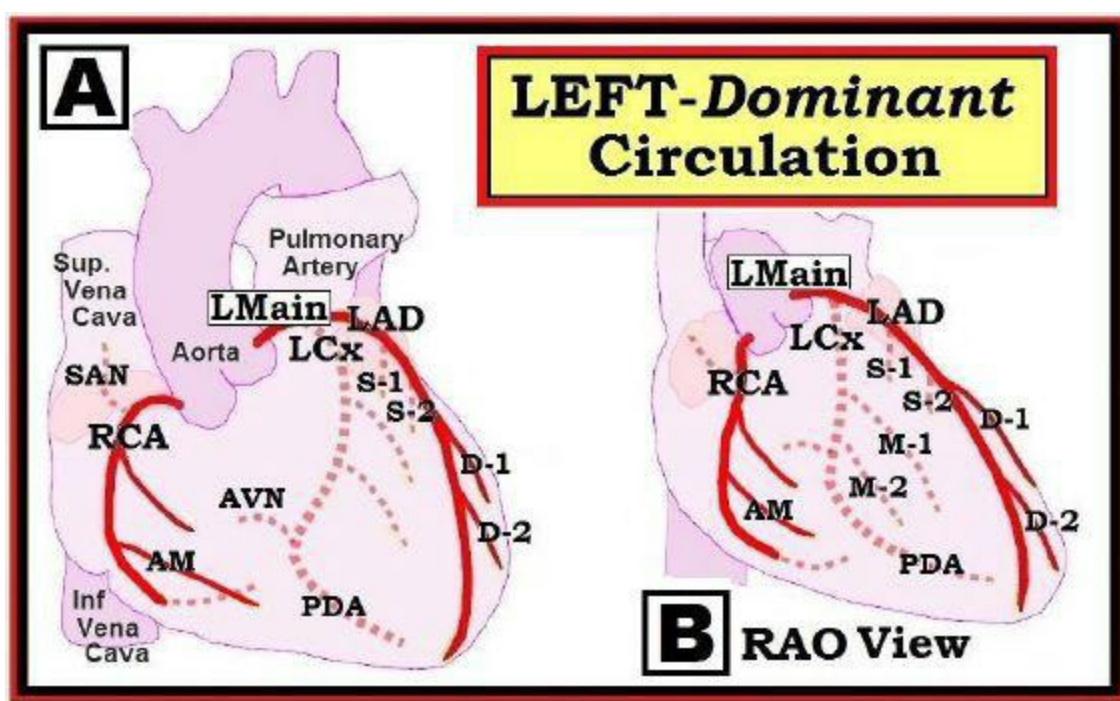


Figure 10.20-1: Schematic depiction of a **left-dominant** circulation (10-20% of patients). The RCA is a smaller vessel. To compensate — the LCx (*Left Circumflex Coronary Artery*) is much **larger** and longer (Compare to Figure 10.18-1). **Panel A** — anterior view. **Panel B** — RAO (*Right-Anterior Oblique*) view (See text). **Abbreviations:** LAD (*Left Anterior Descending Artery*); SAN (*SA Nodal Artery*); AM (*Acute Marginal branches from the RCA*); AVN (*AV Nodal Artery*); PDA (*Posterior Descending Artery*); S-1, S-2 (*Septal Perforator branches*); D-1, D-2 (*Diagonal branches*); M-1, M-2 (*Obtuse Marginal branches from the LCx*).

10.21 – LAD “Wrap-Around”: Taking a Closer Look

In Section 10.18 — We discussed and illustrated the usual distribution of the LAD (*Left Anterior Descending Artery*). Normally, the LAD supplies the *anterior* wall of the heart and the *cardiac* apex. Nevertheless, like *other* major coronary vessels — the LAD is *also* subject to individual variation in its size, course, and the areas of the heart that it vascularizes.

- In some patients — the LAD may terminate *prior* to attaining the apex. In such cases — the apex will be supplied by a larger and **longer-than-usual** PDA arising from either a very dominant RCA — or from a *dominant* LCx vessel (Figure 10.20-1).
- Occasionally — the LAD will be a larger and longer vessel, to the point of extending *beyond* the cardiac apex and “**wrapping around**” to supply the *undersurface* of the heart (**Figure 10.21-1**). At times this extension of the LAD may even serve the function of the PDA. This variant is known as an **LAD “wrap-around”**.
- **Clinical Note** Awareness of the possibility of *acute* LAD occlusion in a patient with this anatomic variant (ie, an *acute “wraparound” LAD lesion*) — explains the ECG pattern of **simultaneous** ST elevation in *inferior* and *anterior* lead areas. Obviously, such infarctions are extensive.
- **Beyond-the-Core:** It is often difficult to assess **acute apical infarction** by ECG — since this area of the heart may *not* be optimally viewed by the standard 12 leads. At other times — notation of *simultaneous* ST elevation in *inferior* and *anterior* leads may suggest acute *apical* involvement. Appreciation of the potential for LAD anatomic variation provides insight to the

various ECG patterns that may be seen. NOTE: Other reasons that may account for *simultaneous* inferior and anterior ST elevation include: **i)** *proximal* RCA occlusion; and **ii)** Takotsubo Cardiomyopathy (*Section 10.61*).

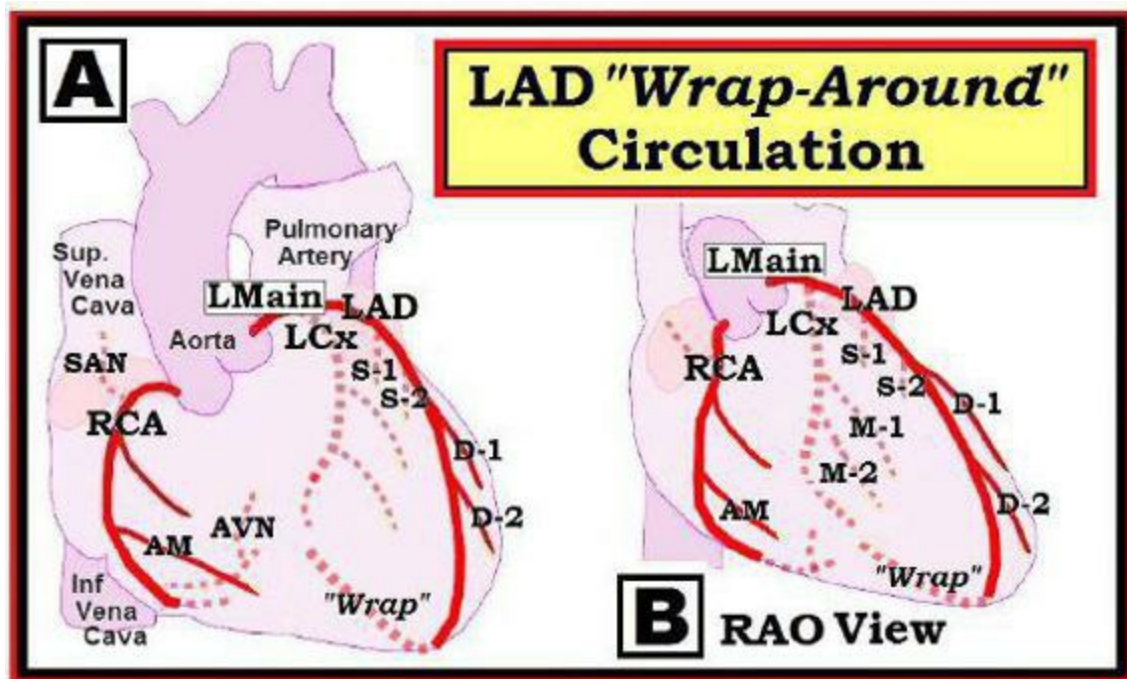


Figure 10.21-1: Schematic depiction of a “wrap around” LAD as an anatomic variant. The RCA is a *smaller* vessel. To compensate — LAD (Left Anterior Descending Artery) will “wrap around” the apex to supply the inferior LV wall (*Compare to Figure 10.19-1*). **Panel A** — *anterior* view. **Panel B** — RAO (Right-Anterior Oblique) view (*See text*). Abbreviations: SAN (SA Nodal Artery); AM (Acute Marginal branches from the RCA); AVN (AV Nodal Artery); S-1,S-2 (Septal Perforator branches); D-1,D-2 (Diagonal branches); M-1,M-2 (Obtuse Marginal branches from the LCx).

10.22 – Identifying the “Culprit” Artery



Assessment of *initial* (and *serial*) ECGs during the *early* course of ACS (*Acute Coronary Syndromes*) may not only suggest the presence of acute (or *impending*) occlusion — but also indicate: **i)** the probable “*culprit*” vessel that is acutely occluded; **ii)** the location (and *extent*) of *evolving* injury; **iii)** associated arrhythmias and conduction disturbances; and **iv)** the *likelihood* that *acute reperfusion* will benefit the patient. The importance of *promptly* assessing the *initial* ECG in a patient with *new-onset* chest pain has *never* been greater.

- **KEY Point:** The *amount* of *benefit* that a patient is likely to derive from *acute* reperfusion of the “*culprit*” (= *infarct-related*) artery is *relative* and depends on both *size* and *acuity* of the infarct. The *more* ST deviation seen (ie, *marked ST elevation* and *reciprocal depression occurring in many leads*) *without* yet forming large Q waves — the *more* the *potential* for benefit.

In these next few sections — We review the *expected* ECG picture for *acute* occlusion of *each* of the major coronary vessels. These include:

- *Acute RCA* (*Right Coronary Artery*) **occlusion** — Section 10.23.
- *Acute LMain* (*Left Main Coronary Artery*) **occlusion** — Section 10.24.
- *Acute LAD* (*Left Anterior Descending Artery*) **occlusion** — Section 10.25.
- *Acute LCx* (*Left Circumflex Artery*) **occlusion** — Section 10.28.

Suggestion: Feel free to refer back to review of the *Coronary Circulation* (in Sections 10.16 through 10.21) — as you contemplate the *expected* ECG picture for *each* of the above entities.

10.23 – Acute RCA Occlusion

As we discussed in Section 10.17 — the *RCA* (*Right Coronary Artery*) serves as a *dominant* vessel in ~85% of patients with a normal coronary circulation. This anatomic picture is *schematically* illustrated in **Panel A** of Figure 10.23-1 — in which the RCA can be seen to supply the RV (*Right Ventricle*) before transitioning to the *PDA* (*Posterior Descending Artery*). The PDA then continues along the *undersurface* of the heart as it supplies the *posterior* and *inferior* walls of the left ventricle (*unlabeled dotted vessel arising from the RCA in Panel A*). Thus, the *RCA* (and its branches) — will normally supply the right ventricle and *inferior plus* posterior walls of the left ventricle.

- In contrast to this picture of a *right-dominant* circulation in Panel A — the remaining ~15% of patients have a *left-dominant* circulation. In this case — it is the *LCx* (*Left Circumflex Artery*) rather than the RCA that supplies the posterior and inferior walls of the left ventricle (**Panel B**).

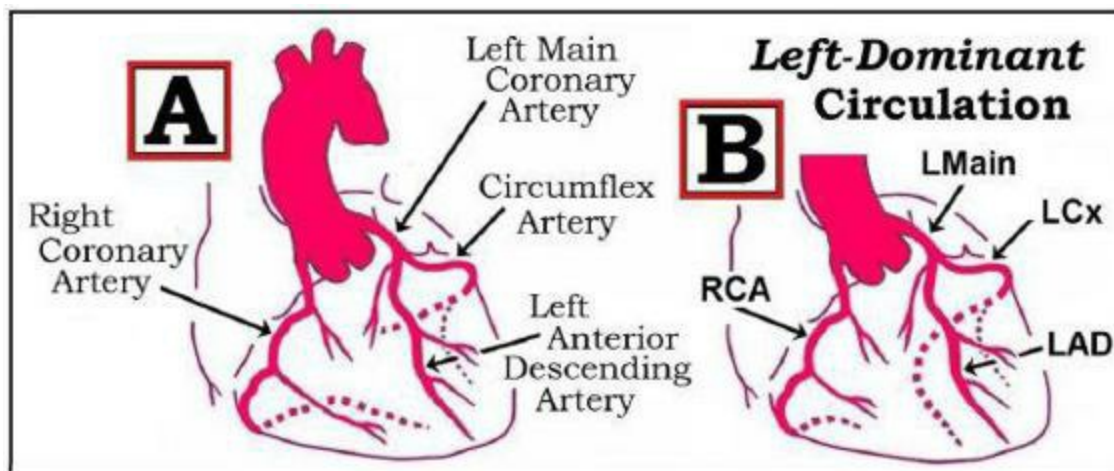


Figure 10.23-1: Comparison of a *right-dominant* circulation (**Panel A**) — which is the most common situation (~85% of patients) vs a *left-dominant* circulation (**Panel B**) — in which the LCx (rather than the RCA) supplies the *posterior* and *inferior* walls of the left ventricle (reproduced from Figure 10.17-1).

ECG findings arising from acute RCA occlusion will vary depending on: **i)** Whether the patient has a *dominant right or left* circulation; **ii)** The relative *site* of occlusion within the RCA (ie, *proximal or more distal occlusion*); **iii)** Any *prior* infarctions that may have occurred; and **iv)** The status of the *collateral* circulation. For simplicity — We describe *expected* ECG findings assuming *no* prior infarctions and *no* alteration in collateral circulation.

- In the 80-90% of patients with a *right-dominant* circulation (**Panel A** in Figure 10.23-1) — the most typical manifestation of **acute RCA occlusion** is ST elevation in all 3 *inferior* leads (II,III,aVF) = **acute inferior MI**.
- **Acute RV (Right Ventricular) MI** — is likely to be seen when there is *proximal* RCA occlusion (Section 10.18). More distal RCA occlusion may spare much of the right ventricle.
- **PEARL: Proximal RCA occlusion** is suggested when **ST elevation** in lead **III** is *more* than in lead **II** (especially if there is **marked ST depression** in lead **aVL**). This picture is seen in **Figure 10.23-2**. In contrast — a *left-dominant* LCx occlusion is suspected when there is *inferior* MI with *less* ST elevation in lead **III** and *less* ST depression in lead **aVL** (especially if there is significant ST elevation in leads **V5,V6**).
- **Posterior MI** — is commonly seen with RCA occlusion (because the RCA most often supplies both *inferior and posterior* walls of the LV). Posterior MI may also be seen with LCx occlusion IF there is a *left-dominant* circulation (Section 10.17). In *either* case — there will *usually* be ECG evidence of **inferior MI** when there is **posterior MI**. **NOTE:** The converse is *not* necessarily true — in that there may be acute inferior MI *without* associated posterior MI. **Beyond-the-Core:** Rarely — *isolated* posterior MI may occur (if the occluded artery is at the level of the PDA). **Bottom Line:** We look for associated acute *posterior* MI when we see ECG evidence of acute *inferior* MI.

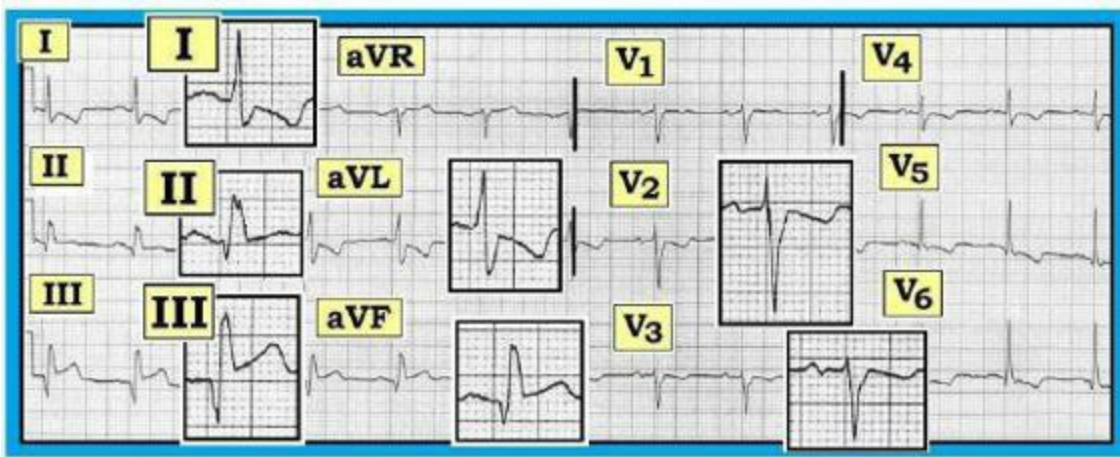


Figure 10.23-2: Acute *infero-postero* MI from **acute proximal RCA occlusion**. Note ST elevation in lead III > II with marked *reciprocal* ST depression in lead aVL. *Posterior* involvement is suggested by the ST depression in lead V2 (*positive “mirror” test*). There may also be acute RV involvement (See text).

Additional Points regarding Acute RCA Occlusion: Keeping in mind the areas of the heart most commonly supplied by the RCA facilitates recognizing acute occlusion of this vessel (Figure 10.23-2):

- **Lead III** is more rightward (*at +120 degrees*) than **lead II** (*at +60 degrees*). As a result — acute occlusion of the RCA generally produces **more ST elevation** in **lead III** than in lead II (Fig. 10.23-2).
- The electrical perspective of lead aVL is virtually *opposite* that of lead III. As a result — the *shape* of the **reciprocal ST depression** in **Lead aVL** often looks like the **mirror-image** of the ST elevation in **lead III**. ST-T wave changes in aVL are typically *marked* when there is *proximal* RCA occlusion — and are generally more prominent than in lead I.
- **Posterior MI** is suggested in Figure 10.23-2 — because of the *positive* “Mirror Test” for lead V2 (ie, *flipping lead V2 over would result in a Q wave with slight-but-real hyperacute ST elevation in this lead*). We discuss the **“Mirror” Test** in more detail beginning in Section 10.33.
- **NOTE:** Associated *posterior* MI may be seen when acute *inferior* MI results from either RCA occlusion or a *left-dominant* LCx occlusion. However, associated **acute RV** (*Right Ventricular*) MI localizes the “culprit artery” to the **RCA** — because the right ventricle is not supplied by the LCx (*Left Circumflex Artery*). We discuss use of **right-sided leads** and the ECG diagnosis of **acute RV MI** in Sections 10.31 and 10.32.
- **Beyond-the-Core:** We suspect that there may also be associated **acute RV MI** in Figure 10.23-2 — because the ST-T wave in lead V1 is relatively flat. Normally, with acute *posterior* MI — there is *similar-appearing* ST-T wave depression in *each* of the anterior leads (*V1, V2, V3*). In contrast, when there is also acute RV MI — *right-sided* ST elevation in lead V1 *cancels out* some of the ST depression that would have been seen from the *posterior* infarction. **Bottom Line:** Suspect associated **acute RV MI** with acute *infero-postero* MI from *proximal* RCA occlusion — IF you see ST segment coving with slight ST elevation in lead V1. The finding of a flat (*instead of depressed*) ST segment in lead V1 (*as is seen in Figure 10.23-2*) — suggests that there may be some canceling out of V1 ST depression by acute RV ST elevation. IF important to

know — **right-sided leads** would help answer this question (*Section 10.31*).

- Clinical Note It is well to remember that **2nd-degree AV Block, Mobitz Type I** (*Section 02.72*) — is most often seen in association with *acute RCA occlusion* (*since the RCA usually supplies the AV nodal artery*). That said — Mobitz I may occasionally be seen with acute LCx occlusion of a *left-dominant* circulation (*in which case the AV nodal artery will usually be supplied by the LCx*).

10.24 – Acute LMain Occlusion

As emphasized in *Section 09.40* — Most patients with **acute LMain** (Left Main Coronary Artery) **occlusion** do not survive. As a result — this entity is not often seen and *unlikely* to be appreciated clinically. Rapid deterioration with patient demise due to cardiogenic shock is the usual result unless acute LMain occlusion can be *immediately* recognized and *immediately* acted on.

- In those *rare* instances when **acute LMain occlusion** is captured on ECG — rather than the picture of *diffuse* ST depression that is characteristic of *severe* LMain disease (*Section 09.40*) — there should be **diffuse precordial ST elevation** in association with **ST elevation** in lead **aVR** (*Figure 10.24-1*).

PEARL: Distinction between *acute LMain* vs *proximal LAD* occlusion may be suggested on ECG by the **relative amount of ST elevation** seen in lead **aVR** *compared to* lead **V1**.

- Think **LMain occlusion** — when ST elevation in lead aVR > V1.
- Think **proximal LAD occlusion** — when ST elevation in lead V1 > aVR.

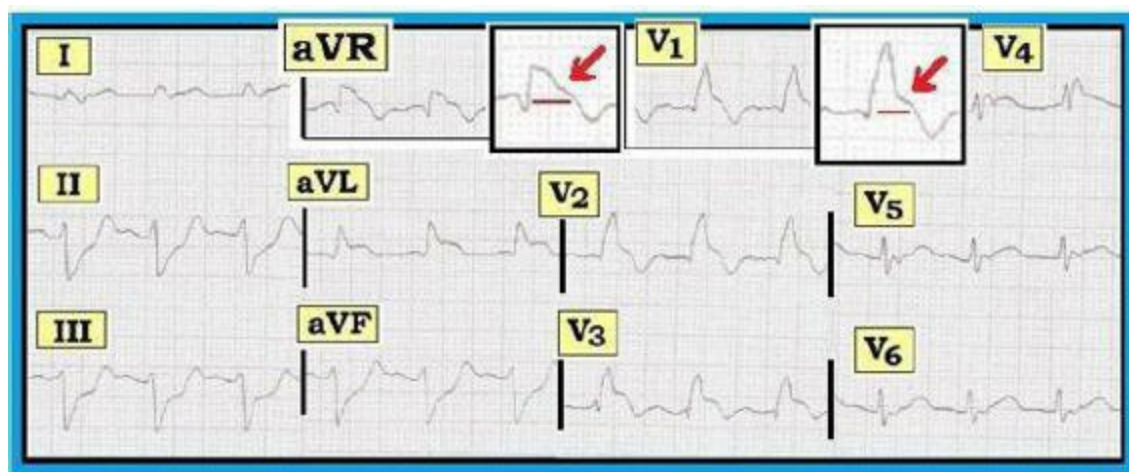


Figure 10.24-1: ECG from an acutely decompensating patient with *new-onset* chest pain (*reproduced from Figure 09.40-2*). New *bifascicular* block (*LAHB/RBBB*) in context with *dramatic* ST-T wave changes seen here (*including ST elevation in lead aVR and in lead V1*) — suggest acute occlusion of *either* the *proximal LAD* or the LMain coronary artery. Given that ST elevation in lead aVR is *at least as much* as in lead V1 (*red arrows*) — We *suspect* acute LMain occlusion (*See text*).

Remember — Most patients with acute LMain occlusion die *before* they reach the hospital. It will therefore be *rare* indeed that you have occasion to evaluate the ECG of such patients. In such rare instances — the clinical presentation will usually be obvious: an *acutely* ill patient with cardiac decompensation from *impending* cardiogenic shock.

- The ECG in **Figure 10.24-1** highlights what to look for: **i)** Conduction defects (*such as RBBB/LAHB that is seen here*); **ii)** Marked ST elevation in precordial leads (*and often also in lead aVL*); **iii)** Marked *reciprocal* ST depression in *other* lead areas; **and iv)** ST elevation in lead aVR that is *at least* as much (*if not more*) than the ST elevation in lead V1.

10.25 – Acute LAD Occlusion

As discussed in Section 10.17 — the **LMain** (Left Main Coronary Artery) is typically a short vessel (10mm) that then **bifurcates** into the **LAD** (Left Anterior Descending Artery) **and** the **LCx** (Left Circumflex Coronary Artery). This anatomic picture is *schematically* illustrated in **Panel A** of **Figure 10.25-1** — in which the **LAD** can be seen to run along the *anterior* epicardial surface of the heart on its path toward the cardiac apex.

- The **LAD** generally supplies: **i)** the *anterior* wall of the heart (*via its diagonal branches*); **ii)** the cardiac apex; **and iii)** a major portion of the conduction system (*via septal perforators that run vertically down through the septum*).

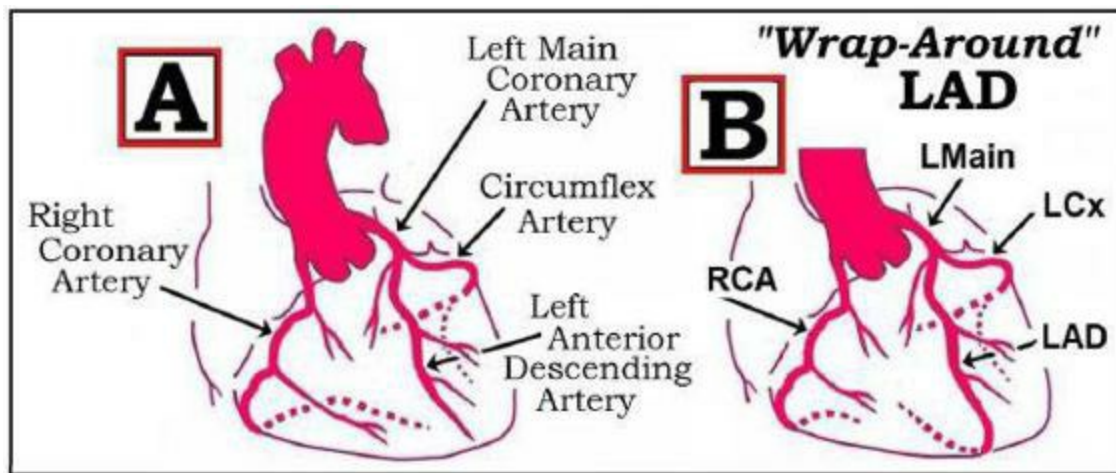


Figure 10.25-1: Comparison of normal LAD circulation (**Panel A**) — *vs* LAD circulation with a “wrap-around” variant (**Panel B**). In this latter situation — the **RCA** is a *smaller* vessel. To compensate — the **LAD** (Left Anterior Descending Artery) will “wrap around” the apex to supply the *inferior* as well as *anterior* wall of the heart (*reproduced from Figure 10.17-1*).

ECG findings arising from **acute LAD occlusion** may vary depending on: **i)** The relative *site* of occlusion within the LAD (ie, *proximal to septal perforators and the 1st diagonal or more distal occlusion*); **ii)** Any *prior* infarctions that may have occurred; **iii)** Presence of any anatomic variants (*such as a “wrap-around” LAD circulation*); **and iv)** The status of the *collateral* circulation. For simplicity — We describe *expected* ECG findings in this Section 10.25 assuming *no* prior

infarctions; *no* alteration in collateral circulation; and *no* anatomic variants.

- In **Section 10.26** — We put forth a *reminder* that not all that elevates ST segments in the *anterior* leads is *anterior* infarction.
- In **Section 10.27** — We discuss the ECG picture of *acute* LAD occlusion when there is an LAD “*wrap-around*” variant (Panel B).

Acute LAD occlusion leads to ***acute anterior MI***. This may be extensive and also involve the *lateral* wall.

- The most typical ECG manifestation of *acute* LAD occlusion is **ST elevation** in *anterior leads* (*usually in ≥ 2 leads between V1-to-V4*).

PEARL: **ST elevation** in **lead aVL** — may provide an *invaluable* clue to the location of the acutely occluded coronary artery. According to a study by Birnbaum et al (*Am Heart J 131:38, 1996*):

- *Suspect acute LAD occlusion proximal* to the **1st Diagonal IF** in *addition* to ST elevation in aVL — there is *also* ST elevation in leads V2-through-V5. This is the most common situation when there is ST elevation in lead aVL.
- *Suspect 1st Diagonal branch occlusion IF* in *addition* to ST elevation in aVL — there is ST elevation in lead V2 (*but not in V3, V4, V5*).
- *Suspect LCx occlusion (especially of the 1st obtuse marginal branch)* — **IF** there is ST elevation in aVL but *not* in lead V2 (*and not in other anterior leads*).

NOTE: *Anterior* ST elevation ***without*** ST elevation in lead aVL — suggests more *distal* LAD occlusion *after* takeoff of the 1st Diagonal.

- **PEARL:** In addition to recognizing ST elevation in lead aVL *with* marked *anterior* ST elevation — there are 2 *additional* ways to identify patients at **high risk** of impending **proximal LAD occlusion**. These are: **i)** Recognition of **Wellens’ Syndrome** (*Section 10.54*); and **ii)** Recognition of **DeWinter T Waves** (*Section 10.57*).

Apply these concepts to the ECG shown in **Figure 10.25-2** — obtained from a patient with *new-onset* chest pain. There is obvious evidence of a **large acute anterior STEMI** (*marked anterior ST elevation*).

- Is the ECG in Figure 10.25-2 likely to indicate *acute* LMain occlusion — or is the “*culprit*” **artery** more likely to be the LAD?
- If you suspect *acute* LAD occlusion — Is the occlusion more likely to be *proximal* or *distal* LAD in location?

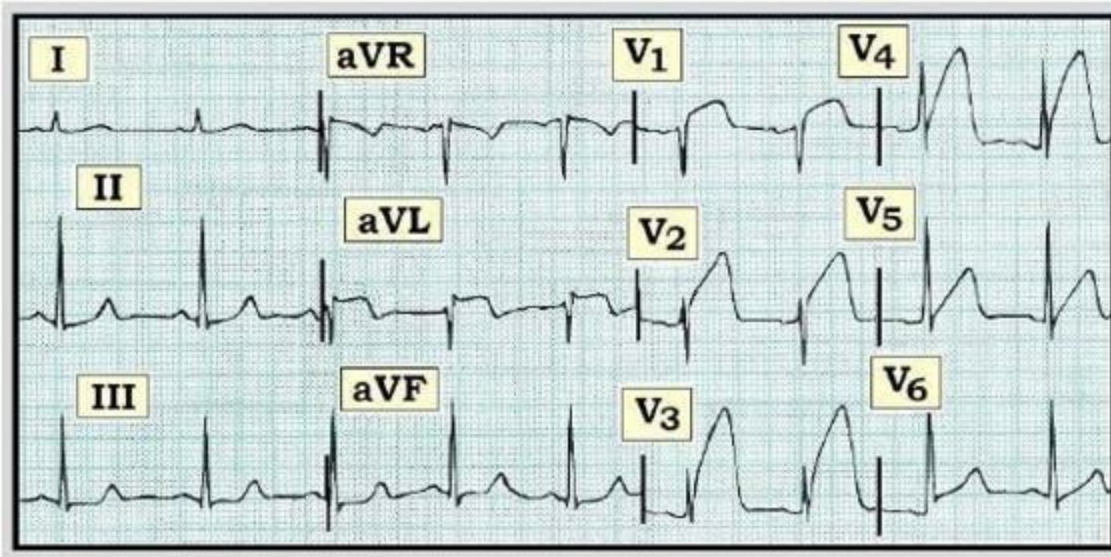


Figure 10.25-2: ECG obtained from a patient with *new-onset* chest pain. An obvious acute *anterior* STEMI is present. What is the likely “culprit” artery? Is the lesion likely to be proximal or distal? (See text).

Answer to Figure 10.25-2: The rhythm is sinus. The most remarkable finding on this ECG is the *marked* ST elevation in leads V1-through-V5. There is also ST elevation in lead aVL. Subtle ST depression is present in the inferior leads as a *reciprocal* change.

- This ECG and the clinical picture is ***not*** suggestive of ***acute LMain occlusion*** because: **i)** Acute LMain occlusion is rare. Most patients die *before* reaching the hospital; **and ii)** **ST elevation** is ***marked*** in lead **V1** ***and*** ***absent*** in lead aVR. This strongly suggests occlusion of the LAD and ***not*** the LMain (Section 10.24).
- We strongly suspect ***acute proximal LAD occlusion*** for the ECG picture shown in Figure 10.25-2. This is because: **i)** Anterior ST elevation is extensive and *marked* in amount; **ii)** There is ***significant ST elevation*** in lead **aVL**; **and iii)** ST elevation is significant in lead **V1** ***and*** ***absent*** in lead aVR.

Additional Clinical Notes Regarding Proximal LAD Occlusion: As noted earlier — the *major* branches of the LAD are: **i)** the ***septal perforators***; **and ii)** one or more ***diagonal branches***.

- Patients with acute *proximal* LAD occlusion (*that occurs before takeoff of the 1st septal perforator*) — are at high risk of conduction system damage (*Mobitz II 2nd-degree AV block; anterior or posterior hemiblock; BBB*).
- ***Beyond-the-Core:*** In addition to the ECG signs of ST elevation in lead aVL ***and*** ST elevation in lead V1 > aVR — the finding of ***new RBBB*** (*especially if a QR pattern is seen in lead V1 instead of an rSR' pattern*) — makes it extremely likely that there is significant septal damage.
- ***NOTE:*** In many ways — a very *proximal* LAD lesion is almost a ***“left-main equivalent”***, in that extent of myocardial damage is ***large*** ***and*** risk of conduction system involvement is high. Acute *proximal* LAD occlusion has been known as a ***“widow-maker”*** lesion — because of the understandably *high mortality* associated with this event. This explains the ***urgency*** of ***recognizing*** the *uncommon* but *foreboding* ECG signs of *Wellens’* syndrome (Section 10.54)

10.26 – Anterior ST Elevation: Not Always an Anterior MI

In the interest of clinical relevance — We feel it important to issue a **reminder** that the ECG finding of **anterior ST elevation** is not always due to acute *anterior* STEMI. **Other Causes** of *anterior* ST elevation that may need to be considered include: **i)** *Early repolarization* (discussed in detail in Section 09.19 and Section 09.23); **ii)** Pericarditis **iii)** LVH (See below in **Figure 10.26-1**); **iv)** LBBB; **v)** Takotsubo; and **vi)** LV aneurysm.

- **Clinical correlation** and comparison with *prior* tracings may sometimes be needed to distinguish such patterns from acute injury.

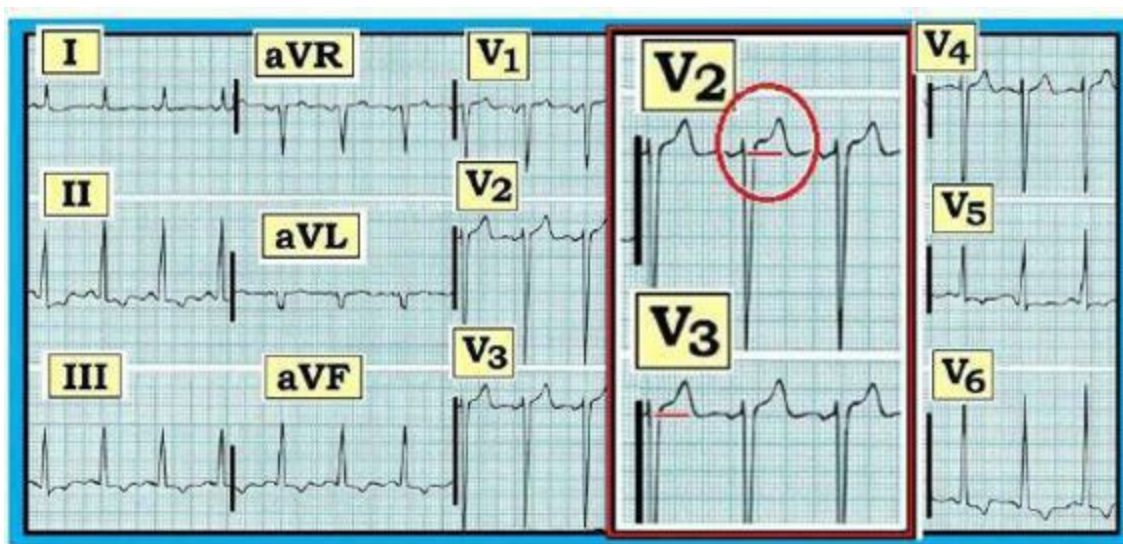


Figure 10.26-1: ECG obtained from a patient with longstanding hypertension — illustrating **ST elevation not due to acute MI** (reproduced from **Figure 08.9-2**). If this patient were to be having “chest pain” at the time this ECG was recorded — it might be easy to misinterpret the several millimeters of ST elevation in leads V2,V3 as indicative of a new event. That said — it is far more likely that the **anterior ST elevation** seen in the *blow-up* insert is an **infarct “mimic”** and merely reflects the *reciprocal* of the LV “strain” pattern seen in lead V6 because: **i)** the dramatic increase in QRS amplitude and typical “strain” ST-T wave morphology in lead V6 “shouts out” that the patient has **LVH** and *not* an MI; **ii)** ST elevation is essentially *limited* to 2 leads; and **iii)** the *shape* of ST elevation in leads V2,V3 is concave up and the *mirror-image* reciprocal of ST-T wave depression in V6 (*vs* ST coving that would be more characteristic of acute injury). That said — it may sometimes be difficult to distinguish between simple LVH vs LVH with *superimposed* acute ST elevation.

10.27 – Acute Occlusion of an LAD “Wrap-Around”

In Sections 10.17 and 10.25 — We discussed normal coronary anatomy of the **LAD** (*Left Anterior Descending Artery*). This anatomic picture is *schematically* illustrated again in **Panel A** of **Figure 10.27-1** — in which the **LAD** is seen passing along the *anterior* epicardial surface of the heart on its path toward the cardiac apex.

- The **LAD** typically supplies: **i)** the *anterior* wall of the heart (*via its **diagonal** branches*); **ii)** the cardiac apex; and **iii)** a major portion of the conduction system (*via **septal perforators** that run vertically down through the septum*).
- Approximately **5-10%** of normal subjects have an anatomic “**wrap-around**” **LAD** variant circulation (**Panel B**). In such cases — the **LAD** is a **larger** and **longer** vessel, to the point of *extending beyond* the cardiac apex and “*wrapping around*” to **supply** the **undersurface** of the heart (*dotted vessel extending underneath the apex from the LAD in **Panel B** of Figure 10.27-1*). In patients with this anatomic variant — the LAD provides the principal blood supply to *both* anterior and inferior walls of the heart.

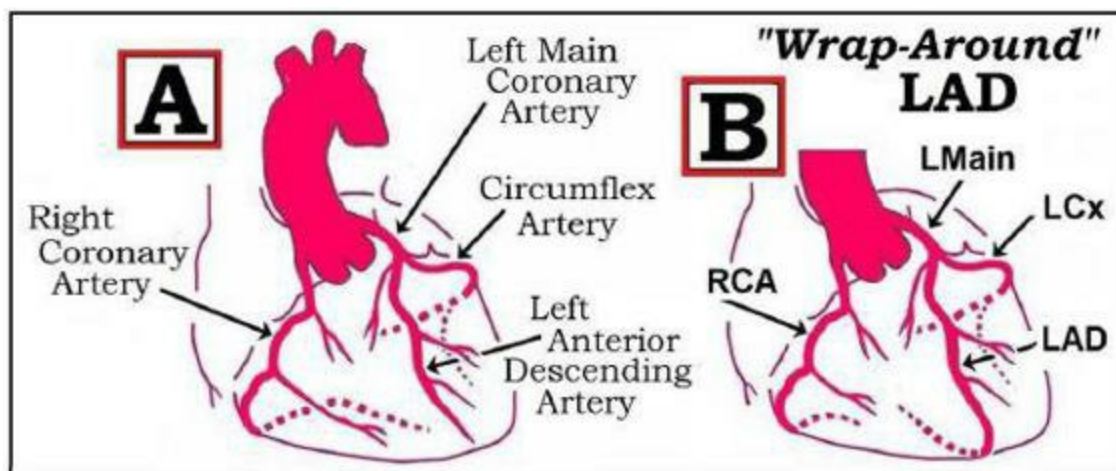


Figure 10.27-1: Comparison of normal LAD circulation (**Panel A**) — vs LAD circulation with a “**wrap-around**” variant (**Panel B**). In this latter situation (*which is seen in ~5-10% of normal subjects*) — the **RCA** is a *smaller* vessel. To compensate — the **LAD** (*Left Anterior Descending Artery*) will “*wrap around*” the apex to supply the *inferior* as well as *anterior* wall of the heart (*reproduced from Figure 10.25-1*).

An ECG picture that has at times been confusing is **simultaneous** presence of **acute ST elevation** in both **anterior** and **inferior** leads. This is because one normally associates acute *inferior* ST elevation with RCA occlusion — whereas acute *anterior* ST elevation is associated with LAD occlusion. A study by Sadanandan et al (*Am Heart J* 146:653-661, 2003) provides insight to *anatomic* explanation of this *unexpected* ECG finding. We integrate findings from the Sandanandan study while emphasizing the following *clinical* points:

- Approximately 15% of patients with acute **anterior** ST elevation also manifest **inferior** ST elevation.
- Surprisingly — about *half* of these patients with acute *anterior* and *inferior* ST elevation are found on cath to have **proximal RCA** rather than LAD occlusion as the “culprit” artery. Presumably — this is a result of RCA marginal branches extending over to supply part of the anterior wall or some other collateralization pattern (*Section 10.18*).
- **ECG Clues** suggesting the “**culprit artery**” is the **proximal RCA** rather than a wrap-around LAD are: **i)** ST elevation in **lead III** > lead II; **ii)** ST elevation in lead V1 > lead V3 (*as one would expect if there was acute RV involvement*); and **iii)** Lack of progression of ST elevation

as one moves from lead V1-to-V4 (*with LAD occlusion ST elevation tends to become more by leads V3,V4*).

- In contrast — a “**wrap-around**” LAD should be suspected as the “**culprit artery**” IF: **i)** ST elevation in **lead II** > lead III; **ii)** There is only *minimal* ST elevation in lead V1; **and iii)** There is *progressive* and *persistent* ST elevation as one moves from lead V1 toward leads V3,V4 (**Figure 10.27-2**).
- Surprisingly — patients with *anterior* STEMI from acute occlusion of a “wrap-around” LAD do not necessarily have large infarctions. This is because the site of acute occlusion is *not* necessarily proximal, but may instead be in the mid- or distal-LAD.
- Beyond-the-Core: Another entity to consider for the presence of acute *simultaneous* inferior and anterior ST elevation is Takotsubo Cardiomyopathy — which may produce an ECG picture *similar* to that shown in *schematic Figure 10.27-2*. Cardiac catheterization is sometimes needed to make this diagnosis (See Section 10.61).

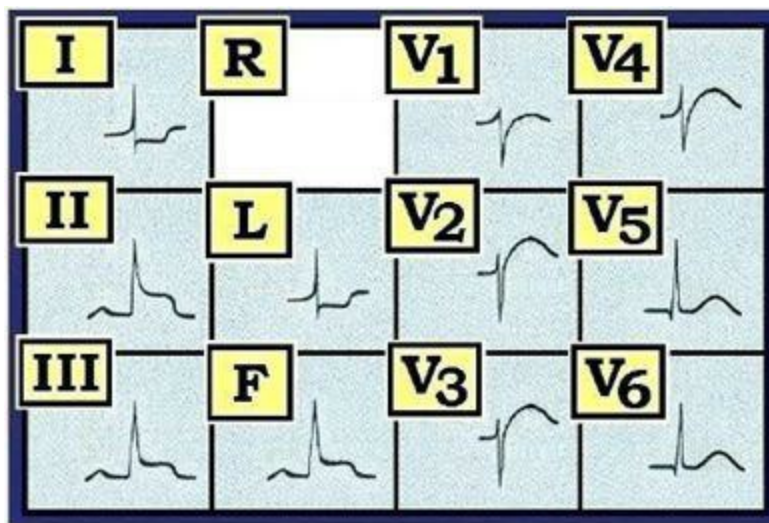


Figure 10.27-2: Schematic ECG with both inferior and anterior ST elevation in a patient with *new-onset* chest pain. The “culprit artery” could be either a *proximal* RCA or *wraparound* LAD lesion. That said — 3 clues that the “culprit” artery is a ‘wraparound’ LAD in this case are: **i)** ST elevation in lead III is less than in lead II; **ii)** ST elevation in V1 is minimal; **and iii)** ST elevation *progresses* and *persistent* as one moves from V1 toward V3,V4 (See text).

10.28 – Acute LCx (Left Circumflex) Occlusion

As emphasized in Section 10.23 — in *most* subjects (~85% of the population) the **RCA** (*Right Coronary Artery*) is a **dominant** vessel. As such — the RCA normally supplies the RV (*Right Ventricle*) on its way as it transitions to the **PDA** (*Posterior Descending Artery*). The PDA then continues along the *undersurface* of the heart as it supplies the *posterior and inferior* walls of the left ventricle (*unlabeled dotted vessel arising from the RCA in Panel A of Figure 10.28-1*).

- The **LCx** (*Circumflex*) **Artery** — wraps around the *lateral* free wall of the LV (*Left Ventricle*). In most patients (ie, when the RCA is dominant) — the LCx becomes a relatively *small* vessel after giving rise to one or more **obtuse marginal branches** that supply the lateral free wall (*dotted vessels arising from the LCx in Panel A*).

- In contrast to the situation in Panel A (in which the RCA is dominant) — between **10-20%** of patients have a **left-dominant circulation** (Panel B). In this case — the RCA is a *smaller* vessel. To compensate — the **LCx** (*Left Circumflex Artery*) is **larger** and gives rise to the **PDA** (*large unlabeled dotted vessel arising from the LCx in Panel B*). In a **left-dominant circulation** the *inferior* and/or *posterior* wall of the LV is supplied by the LCx (Panel B).

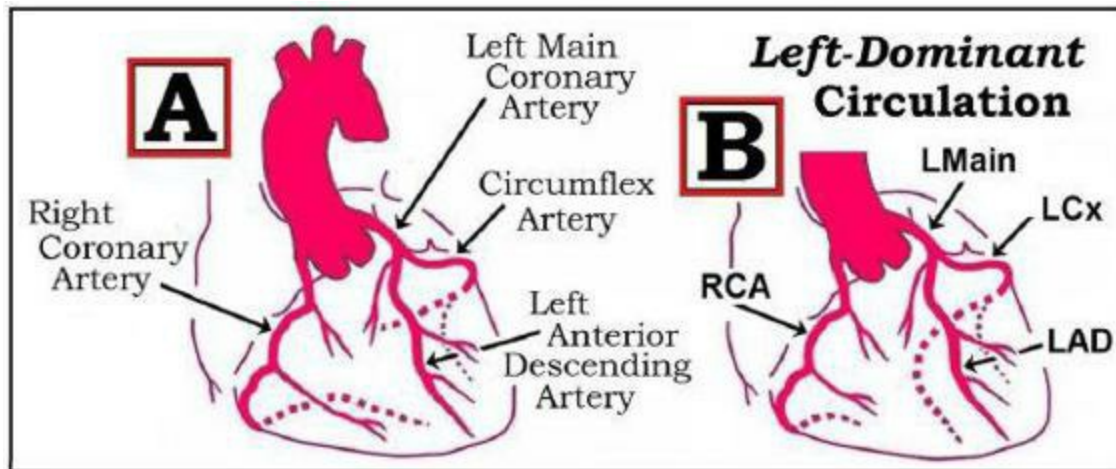


Figure 10.28-1: Comparison of a *right-dominant* circulation (Panel A) — which is the most common situation (~85% of patients) vs a **left-dominant** circulation (Panel B) — in which the LCx (rather than the RCA) supplies the *posterior* and *inferior* walls of the left ventricle (reproduced from Figure 10.23-1).

ECG findings arising from acute LCx occlusion will vary depending on: **i)** Whether the patient has a *dominant* right or left circulation; **ii)** The relative *site* of occlusion within the LCx (ie, *proximal* or *more distal occlusion*); **iii)** Any *prior* infarctions that may have occurred; and **iv)** The status of the *collateral* circulation. For simplicity — We describe *expected* ECG findings assuming *no* prior infarctions and *no* alteration in collateral circulation.

- **Acute occlusion** of the **Circumflex (LCx) artery** leads to **lateral MI**. This is manifest on ECG by ST elevation in one or more of the *lateral* leads.
- Despite assessment of the **LV lateral wall** by *no less* than **5** of the 12 leads (*I, aVL; V4, V5, V6*) — the **high-lateral wall** may not be well visualized. This part of the LV is typically supplied by the **1st Diagonal branch** of the **LAD** (*and not by the LCx*). Thus — **Lead aVL** really provides more information about the site of LAD occlusion (*proximal* or *distal* to the *1st Diagonal branch*) than it does about the LCx (*Section 10.25*).
- The other “*high-lateral*” lead — is **Lead I**. In general — lead I provides *little* independent information regarding the site of acute occlusion. It may be best to think of lead I as providing a *similar* electrical viewpoint as lead aVL, albeit ECG changes in lead I are usually less prominent than in aVL.
- The **lateral leads** that most consistently assess the part of the heart supplied by the LCx are leads V5, V6. Therefore — **ST elevation** in leads **V5, V6** most often suggests **acute LCx occlusion**. *Neighboring lead V4* may manifest similar findings as V5, V6 — but by itself is of *little* help in localizing the site of acute occlusion.

- **NOTE:** Exceptions exist in which ST elevation in leads V5,V6 may not necessarily indicate LCx occlusion. This may occasionally occur in patients with large PLA (*Postero-Lateral Artery*) branches that arise from the PDA branch of the RCA and supply the lateral wall or in patients with an *unusual* pattern of collateral circulation. **Otherwise** — acute ST elevation in leads V5,V6 = **acute LCx occlusion**.

Clinical Synthesis IF there is **ST elevation** in “**high-lateral**” leads (ie, lead I and/or lead aVL) — but there is *no* ST elevation in any of the lateral *precordial* leads (V4,V5,V6) — the site of **acute occlusion** is probably in the **LAD**. This premise will be supported by the presence of ST elevation in one or more of the *anterior* leads (*Section 10.25*).

- **IF** on the *other* hand, there is ST elevation in lead I and/or aVL and ST elevation in lateral *precordial* leads (V4,V5,V6) but not in any *anterior* leads — then **acute LCx occlusion** is the likely “culprit” artery.
- **Beyond-the-Core:** Recently — MRI correlations with cardiac anatomy, coronary artery distribution, and ECG findings suggest that traditional ECG terminology is *not* as accurate as previously thought (*Bayes de Luna et al: Circulation 114:1755, 2006*). Therefore — acute ECG changes *isolated* to the “**high-lateral**” leads (= leads I,aVL) — appear to more accurately reflect acute infarction in the **mid-anterior wall** rather than in the “lateral” wall of the heart. These MRI correlations are consistent with the *Clinical Synthesis* we propose above.

ECG findings differ with **acute LCx occlusion** in a **left-dominant** system. As emphasized in **Panel B** of **Figure 10.28-1** — the LCx is a *larger* vessel in the 10-20% of patients with a *left-dominant* circulation, in which case the LCx supplies the inferior and posterior walls of the LV *as well as* the *lateral* wall.

- **Suspect Circumflex dominance** — **IF** i) you see *infero-postero-lateral* MI; and ii) there is significant ST elevation in leads V5,V6.

Consider the situation in **Figure 10.28-2** — obtained from a patient with *new-onset* chest pain.

- Interpret this ECG. Localize the area(s) of acute infarction.
- Which coronary artery is likely to be acutely occluded?

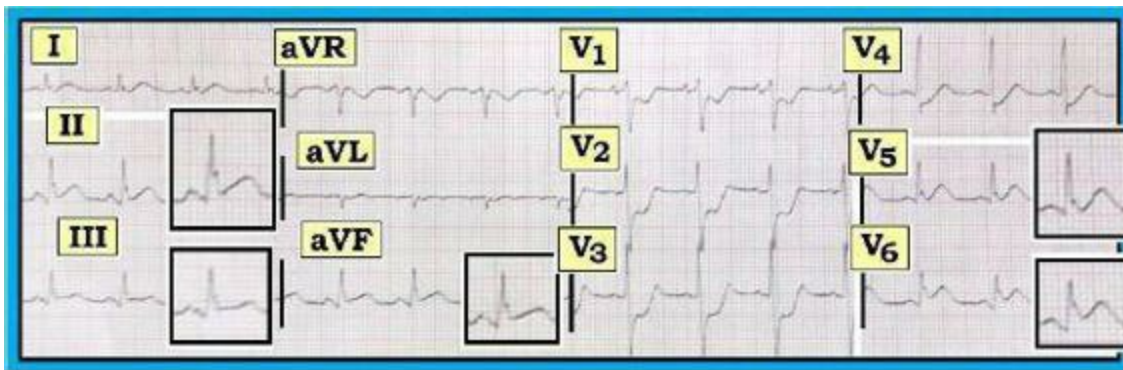


Figure 10.28-2: Acute *infero-postero-lateral* MI. This distribution on ECG suggests a **left-dominant circulation with acute LCx occlusion**. Findings against RCA involvement are: **i)** *Less* ST elevation in lead III *than* lead II; **ii)** *minimal* ST depression in aVL; and **iii)** ST elevation in V5,V6 (See text).

Answer to Figure 10.28-2: The rhythm is sinus. Intervals and axis are normal. No chamber enlargement. Regarding **Q-R-S-T Changes**:

- *Small* q waves are seen in leads II,III,aVF; and V5,V6.
- Transition is normal (*occurs between lead V2-to-V3*) — albeit the R wave is a bit *taller-than-expected* in leads V2,V3.
- There is *subtle-but-real* **ST elevation** in *each* of the **inferior leads** (II,III,aVF) — with suggestion of **hyperacute T waves** (*especially in lead II*). A similar pattern of *slight* J-point ST elevation is seen in **leads V5,V6** — with suggestion of *hyperacute* T waves in these leads.
- There is **marked ST depression** in leads V1-thru-V4.

Impression: Sinus rhythm with **acute infero-postero-lateral STEMI** (ST Elevation Myocardial Infarction). The cath lab should be activated. We suspect **acute LCx** (Left Circumflex Artery) **occlusion** in a *left-dominant* system.

- Although **q waves** in the *infero-lateral* leads are small and narrow — these are the very *same* leads that manifest ST elevation with *hyperacute* T waves. While these might possibly be normal *septal* q waves — it is far more likely that they reflect ongoing *acute* infarction. In *any* case — We should know the answer shortly (*if the q waves become larger as the infarct evolves* — or — *if they resolve after reperfusion*).
- The ECG picture in leads V1,V2,V3 strongly suggests *associated* **acute posterior infarction**. We say this despite the reality that *none* of the standard 12 leads directly visualize the *posterior* wall of the LV (Left Ventricle). As will be discussed in Section 10.35 — **to diagnose acute posterior MI**, one either has to: **i)** Obtain **additional leads** that directly visualize the posterior wall (= *leads V7,V8,V9*); or **ii)** Perform a “**mirror**” test (*turning the tracing over and holding it up to the light* — which provides a mirror-image view of the anterior leads — **Figure 10.28-3**).

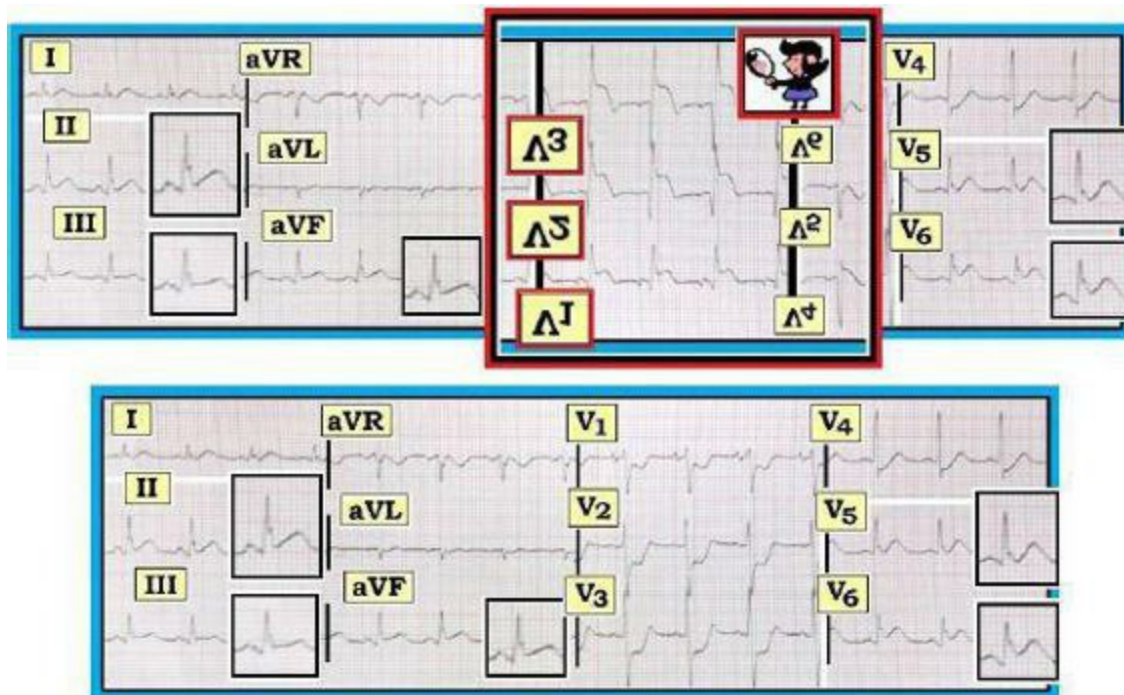


Figure 10.28-3: Diagnosis of *acute posterior* MI is made from Figure 10.28-2 by applying the “*mirror*” test (ie, turning the tracing over and holding it up to the light). Doing so (*TOP* tracing) shows the *anterior* ST depression and relatively tall R waves in V2,V3 of the *LOWER* tracing *become* Q waves and ST elevation when the tracing is flipped over (See within the red rectangle of the *TOP* tracing).

Figure 10.28-2: Localizing the “Culprit” Artery — Given the presence of acute *inferior* infarction without *anterior* ST elevation in Figure 10.28-2 — the “culprit” artery is almost certainly either the RCA (Right Coronary Artery) or the LCx (Left Circumflex Coronary Artery).

- As discussed in Section 10.23 — **acute proximal RCA occlusion** would be suggested by: **i)** ST elevation in **lead III** > lead II; **ii)** marked *reciprocal* ST depression in lead aVL that is *more* than the ST depression in lead I; and **iii)** ECG evidence of *acute RV* MI (*the LCx does not supply the right ventricle*). While on occasion *acute* RCA occlusion may produce ST elevation in leads V5,V6 (ie, *if there is a large posterolateral branch arising from the PDA branch of the RCA*) — ST elevation in lead V6 should not be more than in lead III.
- In **Figure 10.28.2** (reproduced in the *LOWER* tracing of Fig. 10.28-3) — We strongly suspect the “culprit” artery is the LCx in a **left-dominant** system because: **i)** ST elevation in lead III is *less* than in lead II; **ii)** There is *no* ST elevation in lead aVL; **iii)** There is *no* hint of acute RV involvement (*ST depression is prominent in V1 — whereas we’d expect either a flat or coved ST segment with slight elevation in V1 if there was associated RV involvement*); and **iv)** There is ST elevation in V5,V6 — and this ST elevation in lead V6 is clearly more than it is in lead III.



10.30 – Acute RV MI: *Hemodynamics*

A substantial proportion of patients with acute *inferior* MI also manifest RV involvement. This is especially true when there is **proximal RCA occlusion** — as suggested by the **ECG signs** of: **i)** ST elevation in **lead III** > lead II; and **ii)** *marked* ST depression in lead aVL that is more than in lead I (*Section 10.23*).

- The importance of recognizing RV involvement with *inferior* MI relates to its **different hemodynamics** (*nitroglycerin not advised because it may excessively lower BP; volume-dependent hypotension that often responds well to IV fluid infusion; frequent need for extremely close ICU monitoring*).
- Many patients with acute *inferior* MI from RCA occlusion have at least **some degree of RV involvement**. This should *not* be unexpected — given normal distribution of the RCA that typically supplies *both* the RV *as well as* the *inferior* wall of the LV. That said, even when there is *some* RV involvement — LV involvement often predominates. The **KEY** is to identify those patients at **high risk of decompensation** from altered hemodynamics. Clinically — this may be suggested by: **i)** ECG evidence of a **large acute RV MI** (*Section 10.32*); **ii)** The combination of *inferior* MI with borderline or low BP — especially if signs of *left-sided* heart failure (ie, *rales*) are absent; and **iii)** A *hypotensive* response to nitroglycerin — especially in patients with acute *inferior* MI.
- **Clinical NOTE:** Some emergency systems eliminate all use of NTG (*nitroglycerin*) in any patient with acute *inferior* MI. While realizing the rationale for this practice (*possibility of associated RV involvement with risk of inducing hypotension*) — global elimination of NTG for all patients with *inferior* MI is not necessarily needed or advisable. **BOTTOM Line:** We suggest the following: **i)** Do not use NTG in *acute* MI patients when BP is low (*or relatively low*) — especially when hypovolemia is suspected; **ii)** Be especially cautious in patients suspected of having *significant* RV involvement; **iii)** Careful titration of IV NTG may be indicated in selected patients with adequate BP. In contrast — use of *sublingual* NTG in such patients may be *ill-advised* because of inability to take a sublingual dose back; and **iv)** You should follow the *standard-of-care* protocols put forth at your institution.

10.31 – Acute RV MI: *Use of Right-Sided Leads*

Definitive ECG diagnosis of **acute RV** (*Right Ventricular*) **MI** is made by use of **right-sided ECG leads**. A *right-sided* ECG is performed using *similar* anatomic placement of precordial leads — except that chest leads are positioned on the *right* side of the chest (**Figure 10.31-1**).

- *Right-sided* ECG leads are labeled V1R, V2R, V3R, V4R, V5R, V6R.

- When obtaining leads V1R and V2R — one simply *reverses* the anatomic lead position of normal leads V1 and V2. That is — **normally placed lead V1** will be the **equivalent of right-sided lead V2 (V2R)**. Similarly — normally placed lead V2 will be the equivalent of *right-sided* lead V1 (V1R). This explains why **normally-placed lead V1** is really a **right-sided** lead (Section 10.32).

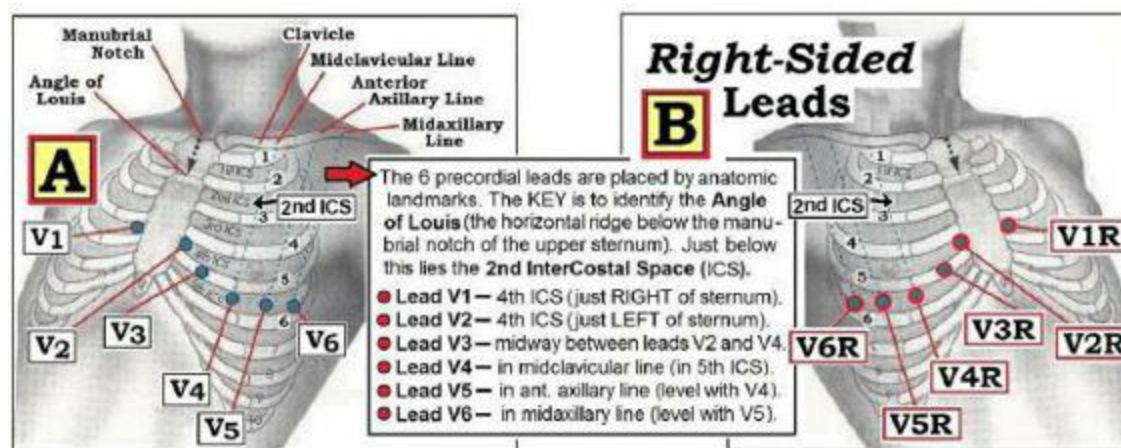


Figure 10.31-1: Anatomic landmarks for normal (*left-sided*) precordial lead placement (**Panel A** — which was previously discussed in Section 03.6). To obtain **right-sided** precordial leads (**Panel B**) — similar anatomic landmarks are used, but leads are placed on the *right* side of the chest (See text).

10.32 – Acute RV MI: Making the Diagnosis by ECG

As stated — ECG diagnosis of **acute RV (Right Ventricular) MI** is made by use of **right-sided ECG leads**. The following findings should be looked for:

- ECG evidence of **associated proximal RCA occlusion** (ie, **acute inferior MI** with *ST elevation in lead III > II* and *marked ST depression in lead aVL*).
- Progressively* increasing ST elevation as one moves across *right-sided* leads. *Right-sided* ST elevation is often **maximal** in lead V4R.
- ST segment coving or straightening (*especially if there is slight ST elevation*) in lead V1 — in association with acute *infero-postero* MI (**Figure 10.32-1**).

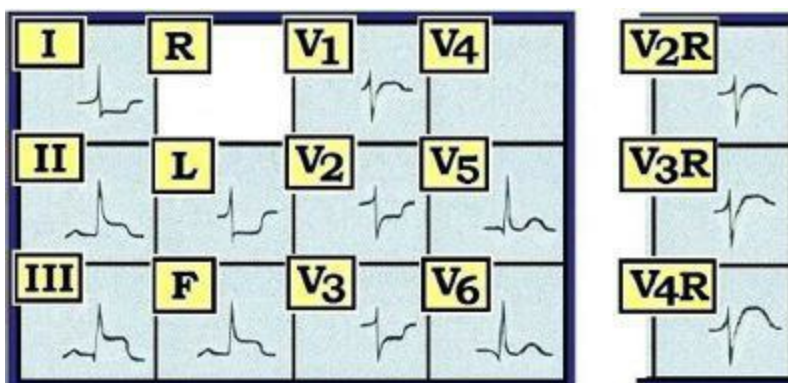


Figure 10.32-1: Schematic ECG showing acute *infero-postero* MI. **Proximal RCA occlusion** is suggested by ST elevation in lead III > lead II with marked *reciprocal* ST depression in lead aVL that

is more than in lead I. **Acute RV MI** is *suggested* by ST coving in lead V1 and *confirmed* by obtaining *right-sided* leads (Note ST elevation in V2R through V4R).

KEY Point: Use of Lead V1 in Figure 10.32-1: Although *right-sided* leads V2R,V3R,V4R confirm the diagnosis of acute RV MI in Figure 10.32-1 — these extra leads are not essential for making the diagnosis:

- As illustrated in Figure 10.31-1 — **lead V1** is really a ***right-sided* lead**. Anatomic landmarks for placement of lead V1 are *the same* as they are for placement of lead V2R.
- The *schematic* ECG in Fig. 10.32-1 shows **acute infero-postero MI**. Acute *posterior* involvement is suggested by the *shape* of ST depression in leads V2,V3 (= *positive “mirror test”* — *as will be discussed in Section 10.35*). When there is simply *posterior* infarction — **lead V1** will manifest a *similar* shape of ST depression as is seen in leads V2,V3. However, IF there is also acute RV MI — then the ST depression *that should have been seen* in lead V1 (*as a result of the posterior MI*) — will be *cancelled out* by the ST elevation in lead V1 from the *acute RV MI*. **BOTTOM Line:** The fact that the ST segment in **lead V1** of *schematic Figure 10.32-1* is coved (*if not slightly elevated instead of being depressed*) — strongly suggests that *in addition* to acute *infero-postero* MI, there is also **acute RV MI**. This is different than the situation with acute *anterior* MI — in which case *not only* lead V1, but *also* other anterior leads will manifest ST elevation (V2,V3,V4).

Clinical Notes: Beyond-the-Core: Opinions vary as to the need for *additional* leads to make the diagnosis of *acute RV MI*. Depending on the ECG recording system used and experience of the person recording the 12-lead tracing — a certain amount of *additional* time will be required to obtain more than the standard 12 leads. This time may be minimal (*seconds*) — or it may take a *precious* minute or more. *You will not know how long it takes to get right-sided lead(s) unless and until you actually time yourself under real-life circumstances.*

- **Our Bias:** We prefer to ***start*** with **12 leads**. Much (*most*) of the time — 12 leads are all that will be needed in order to determine what you need to know to optimally manage the patient. That said — We fully acknowledge that there are times when additional leads may be extremely helpful.
- Clearly, a balance must be struck between *urgency* of the situation — *How Long* it will take to obtain additional leads — and whether this time is really worth what you'll *actually* learn from getting more leads. For example — IF there is obvious acute *proximal* RCA occlusion on *initial* ECG obtained by EMS providers in a *prehospital* setting — there is little to learn that will *change* management by spending extra time prior to transport in recording *right-sided* leads (ie, *the cath lab will need to be activated regardless*).
- In a more stable setting when there is time to obtain *additional* leads — the most *sensitive* ECG indicator of **acute RV MI** is the presence of **≥ 1 mm ST elevation in lead V4R**. This finding may be transient — so ***right-sided* leads** should be obtained *as early as feasible* when looking for RV MI.

Consider the situation in **Figure 10.32-2** — obtained from a patient with *new-onset* chest pain. Note that **right-sided leads** = **V3R** and **V4R** have been obtained (*and are seen instead of leads V3, V4 on this 12-lead tracing*).

- Localize the area(s) of acute infarction.
- What is the location of acute coronary occlusion?

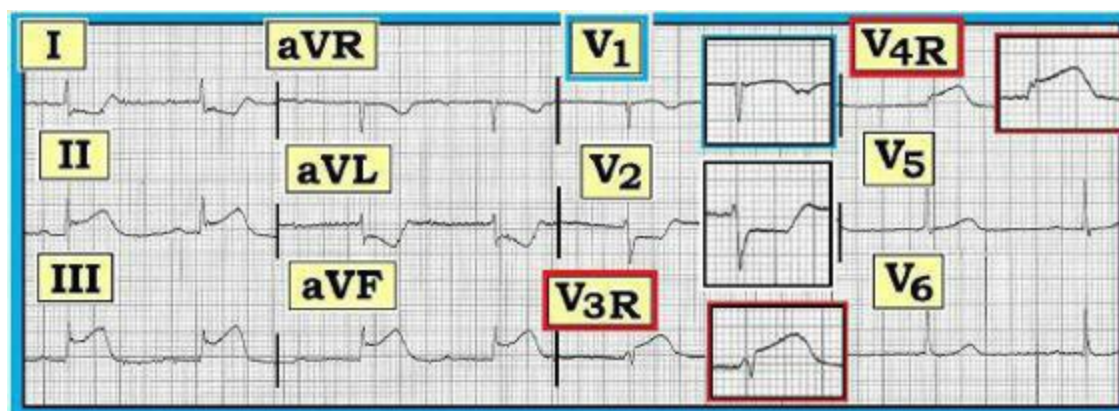


Figure 10.32-2: Acute *infero-postero* MI. In addition — ST coving with slight elevation in lead **V1** (*blue blow-up insert*) suggests **acute RV MI**. This is supported by the presence of *marked* ST elevation in **right-sided leads V3R and V4R** (*red blow-up inserts*). Posterior MI is diagnosed by the *positive* “mirror” test in lead V2 (*See text*).

Answer to Figure 10.32-2: The rhythm is sinus bradycardia. There is *marked* ST elevation in each of the *inferior* leads. ST elevation in lead **III** > lead **II** — suggesting **acute proximal RCA occlusion**. This is supported by the finding of *marked* ST depression in lead **aVL** that is *more* than in lead **I**.

- **Acute posterior MI** — is suggested by the shape of ST depression in lead V2 (*positive “mirror” test*).
- **Acute RV MI** is suggested by ST segment coving (*with a hint of ST elevation*) in lead **V1** in the presence of ST depression in lead V2.
- Use of **right-sided leads V3R and V4R** confirms the diagnosis of **acute RV MI**. Note how *marked* ST elevation is in leads V3R and V4R. Given the presence of sinus bradycardia — *marked* inferior ST elevation with equally *marked* reciprocal ST depression — and large associated *acute* RV MI — this patient is likely to manifest *volume-dependent* hemodynamics, and will clearly require intensive care monitoring.

10.33 – Posterior MI: Use of the Mirror Test



One area of the heart that is not well visualized by any of the 12-leads of a standard ECG is the *posterior* wall of the left ventricle. This clinical reality makes it challenging to recognize new or old *posterior* infarction.

- Use of the “**Mirror**” Test facilitates recognizing ECG changes of **acute posterior MI** from the standard 12-lead tracing (**Figure 10.33-1**).

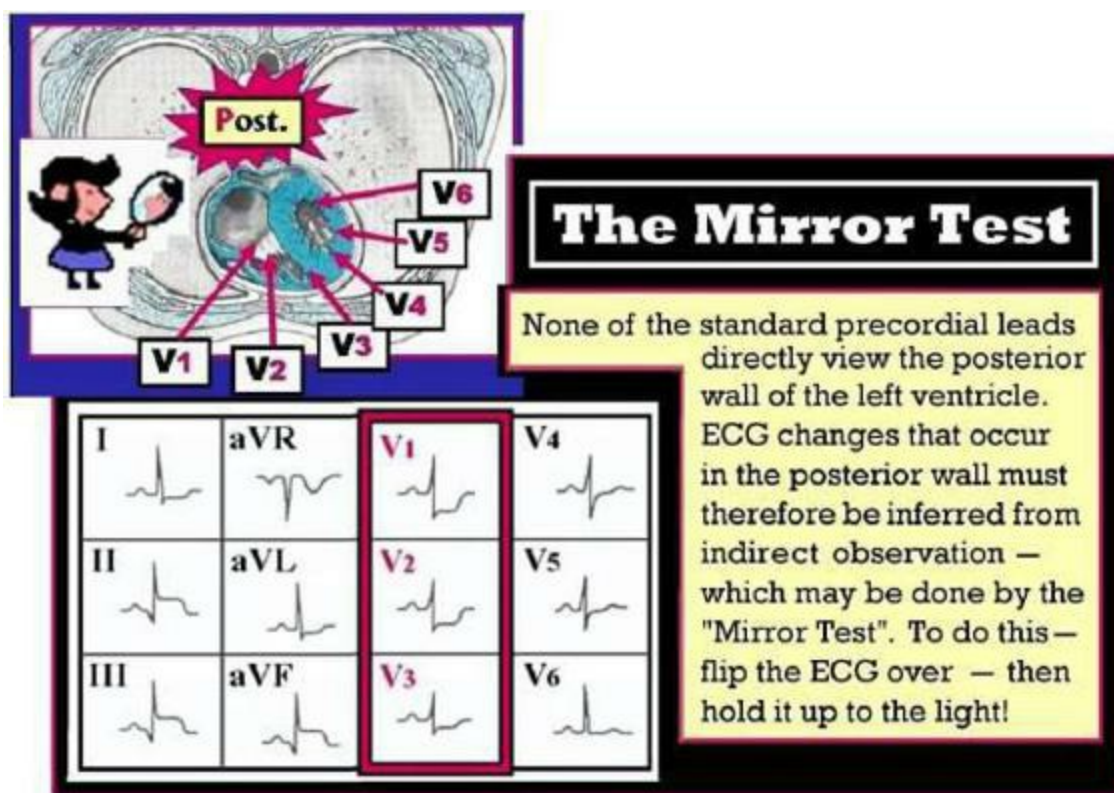


Figure 10.33-1: The “**Mirror**” Test. None of the standard 12 leads of an ECG directly visualize the posterior wall of the left ventricle. As a result — *indirect* visualization of the **opposite** (= *anterior*) wall may be used to provide insight regarding *acute* posterior changes that may be ongoing. **Anterior** leads V1,V2,V3 (within the red rectangle) — are used in the “*mirror*” test (See text).

10.34 – BEYOND-the-Core: Is there Truly a Posterior Wall?

Recently — MRI correlations with cardiac anatomy, coronary artery distribution, and ECG findings suggest that *traditional* ECG terminology is *not* as accurate as previously thought (*Bayes de Luna et al: Circulation 114:1755, 2006*). Thus, the anatomic relationship of the “posterior” wall — is in reality not as directly posterior as depicted in [Figure 10.33-1](#). Instead — what traditionally has been thought of as “posterior” wall involvement is more accurately referred to as involvement of part of the *lateral* LV wall.

- A new, more anatomically accurate terminology has been proposed. This would *change*

reference to the *posterior* LV wall to the ***lateral wall*** instead.

- **MY BIAS:** Realizing the potential *tremendous* benefit MRI correlations may provide toward more accurate anatomic localization — ***traditional ECG terminology*** appears *entrenched* at the current time. Rather than confusing the issue with a novel ECG terminology that is *not yet* in general use — **We favor continued use** of the term ***posterior infarction*** (*with continued distinction between lateral vs posterior walls of the heart*). We devote Sections 10.33 through 10.37 to recognition of *posterior* wall MI according to the *traditional* terminology that we use throughout this ePub. We realize that at some point in the future this terminology may change.

10.35 – FIGURE 10.35-1: *Applying the Mirror Test*

We illustrate application of the “Mirror” Test in *schematic* **Figure 10.35-1**. The test is based on the principle that *anterior* leads provide a *mirror image* of electrical activity occurring over the *posterior* wall of the LV. We emphasize the following points:

- Most patients with acute *posterior* MI — *also* manifest ECG evidence of ***acute inferior MI***. This is seen in the *lower* tracing (**Panel B**) of **Figure 10.35-1** — in which there are small q waves and ST segment elevation in *each* of the *inferior* leads (*II,III,aVF*). In support of the diagnosis of acute *inferior* infarction — is *reciprocal* ST depression in leads I and aVL.
- One looks for a ***tall R wave*** in lead ***V1*** (*or at least taller-than-expected R waves in V2, V3*) plus a *peculiar* shape of ***anterior ST depression***. This is well seen in leads V1,V2,V3 *within* the *red rectangle* in **Panel B**.
- Flipping the tracing over and holding it up to the light (ie, *performing the “mirror” test*) reveals that what was ST depression and a disproportionately *tall* R wave in *anterior* leads (*in Panel B*) — *becomes* a Q wave with worrisome ST elevation = a ***positive “mirror” test*** (**Panel A**). This strongly suggests that in addition to acute *inferior* MI — there is *also* ongoing ***acute posterior MI***.

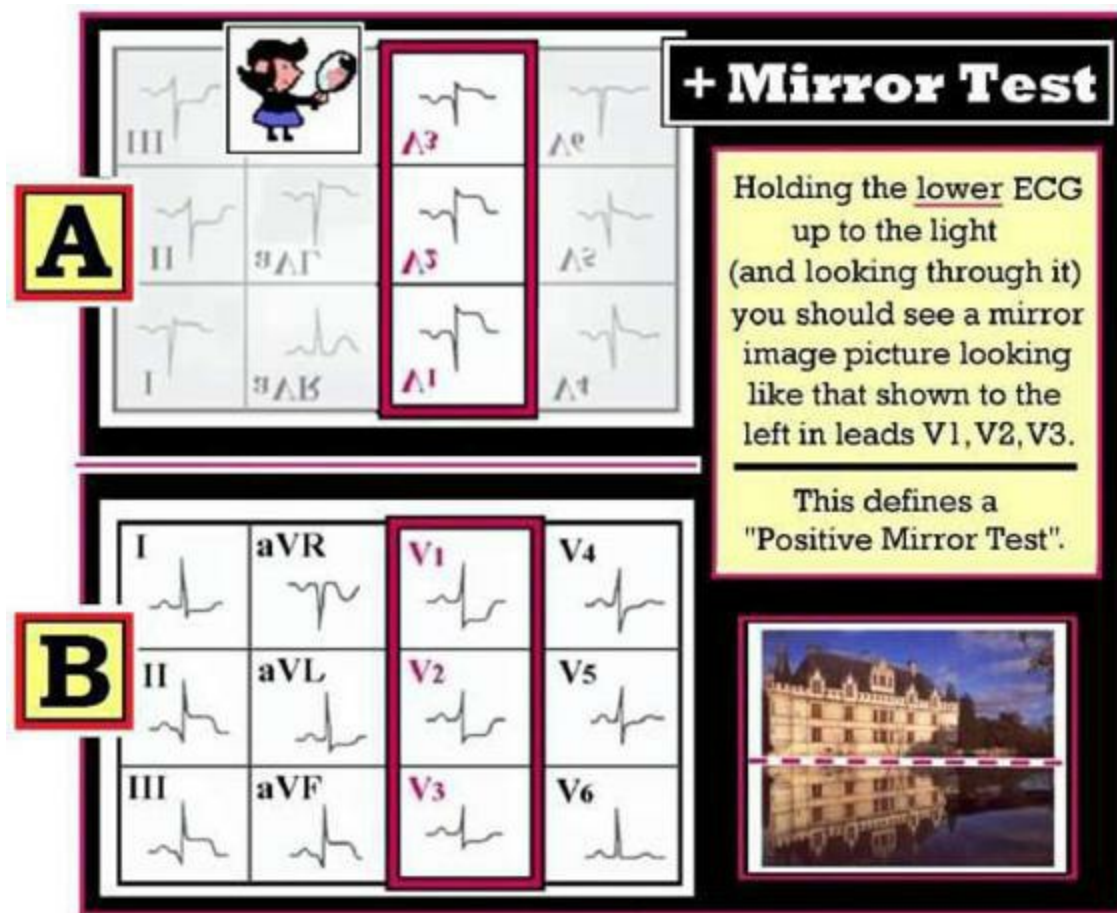


Figure 10.35-1: Positive “mirror test” — as seen when the upright initial ECG in Panel B is *flipped over* (Panel A) and held up to the light. **NOTE:** The *more* ST depression you see in *anterior* leads V1, V2, V3 (Panel B) — the *more* extensive the ***infero-postero* MI** is likely to be!

NOTE: The “mirror” test — is nothing more than a visual aid that we have conceived to facilitate recognition of *posterior* infarction (Grauer K, circa 1983). We have already seen application of this ***visual aid*** to facilitate recognition of the *peculiar* shape of *anterior* ST depression in the example of acute *infero-postero-lateral* MI that was presented in Figure 10.28-3 — which we reproduce below in Figure 10.35-2.

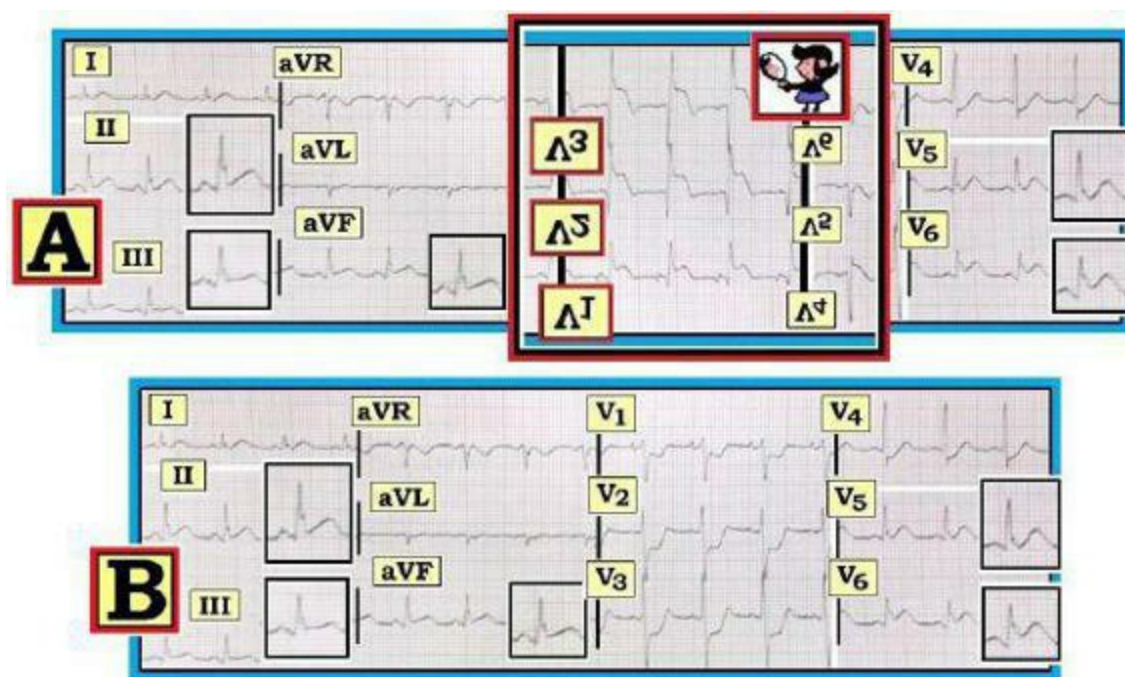


Figure 10.35-2: Illustration of a **positive “mirror” test** in this patient with acute *infero-lateral-postero* MI due to acute occlusion of a *dominant* LCx (reproduced from Figure 10.28-3). The original *upright* ECG is shown in the *lower* tracing (**Panel B**). Note ST elevation in leads II,III,aVF and in leads V5,V6 — consistent with acute *infero-lateral* MI. Diagnosis of associated **acute posterior MI** is made by applying the **“mirror” test** (ie, turning the tracing in Panel B over, and holding it up to the light). Doing so (as shown for leads V1,V2,V3 within the red rectangle in **Panel A**) — reveals how the *anterior* ST depression and relatively tall R waves in leads V2,V3 of Panel B become Q waves and ST elevation when the tracing is flipped over.

10.36 – FIGURE 10.36-1: *Anatomic Landmarks for Posterior Leads*

An alternative approach to using the *mirror* test — is the use of **posterior leads** to diagnose or confirm diagnosis of *posterior* MI. These may be obtained separately — incorporated into a 15-lead ECG (which typically adds leads V4R, V8, and V9 to the standard 12 leads) — or substituted for one or more lateral precordial leads on a standard ECG.

- **Posterior leads** most commonly include leads V7, V8, and/or V9. These leads are obtained by continuing *laterally* along the **same horizontal level** as lead V6 in the *posterior* axillary line (= **lead V7**) — at the *tip* of the left scapula in the back (= **lead V8**) — and in the left paraspinal area *in between* the scapula and posterior spine (= **lead V9**).
- **Anatomic landmarks** for *posterior* leads are shown in **Figure 10.36-1**. We then conclude this segment on *posterior* infarction by illustrating how posterior leads are used to confirm acute posterior MI in Section 10.37.

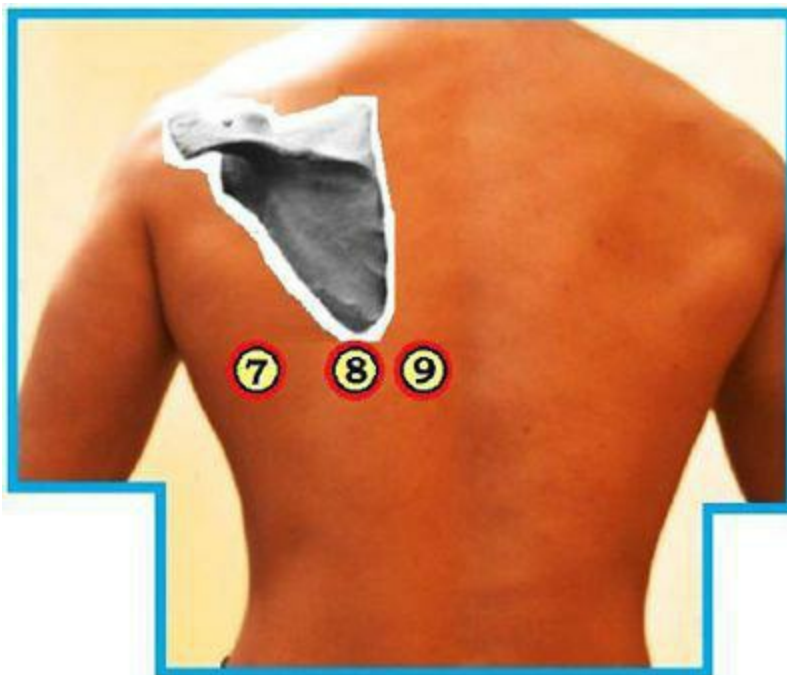


Figure 10.36-1: *Anatomic landmarks for posterior leads*. The **Lead V7** electrode — is placed in the *posterior* axillary line at the *same* horizontal level as lead V6. **Lead V8** — is placed in the back at the tip of the left scapula (*at the same horizontal level as lead V6*). **Lead V9** — is placed in the back in the left paraspinal area *in between* the scapula and posterior spine (*at the same horizontal level as lead V6*).

10.37 – FIGURE 10.37-1: *Isolated Posterior Infarction*

As emphasized in Section 10.35 — *most* patients with acute *posterior* MI *also* manifest ECG evidence of acute *inferior* MI. This is because the RCA commonly supplies *both* the *inferior* *and* *posterior* walls of the left ventricle. Acute occlusion of the RCA therefore commonly results in acute *infero-postero* MI.

- Even when there is *acute* occlusion of a *dominant* LCx artery (*as in Panel B of Figure 10.35-2*) — acute *inferior* *and* *posterior* infarction almost always occur together.
- On occasion, however — ***posterior MI*** may be ***isolated***. This may occur IF the acute occlusion is at the level of the PDA (*Posterior Descending Artery*). Because the PDA may arise from *either* the RCA or the LCx (*if there is a left-dominant circulation — as described in Section 10.28*) — it may *not* be possible by ECG to identify the “culprit artery” on those relatively rare occasions when there is *isolated* posterior MI.

Consider the ECG shown in Panel A of **Figure 10.37-1** — in which there is ECG evidence of ***isolated acute posterior MI***. Note the *absence* of any indication of acute inferior MI (*no inferior ST elevation — and no more than minimal if any ST depression in leads I and aVL*).

- *Acute* changes in Panel A of Figure 10.37-1 are subtle. Nevertheless — the R wave in leads V2,V3 of Panel A are disproportionately tall (*especially in view of no more than a tiny r wave in lead V1*) — and there is *significant* ST depression that is most *marked* in leads V2,V3,V4. ***Acute posterior MI*** is strongly suggested by a *positive* “mirror” test (**Panel C** compared to upright leads V1,V2,V3 in Panel B).
- Acute *posterior* MI is ***confirmed*** by Q waves with ST elevation in ***posterior*** leads V7 and V9 (*leads outlined in red in **Panel D***).

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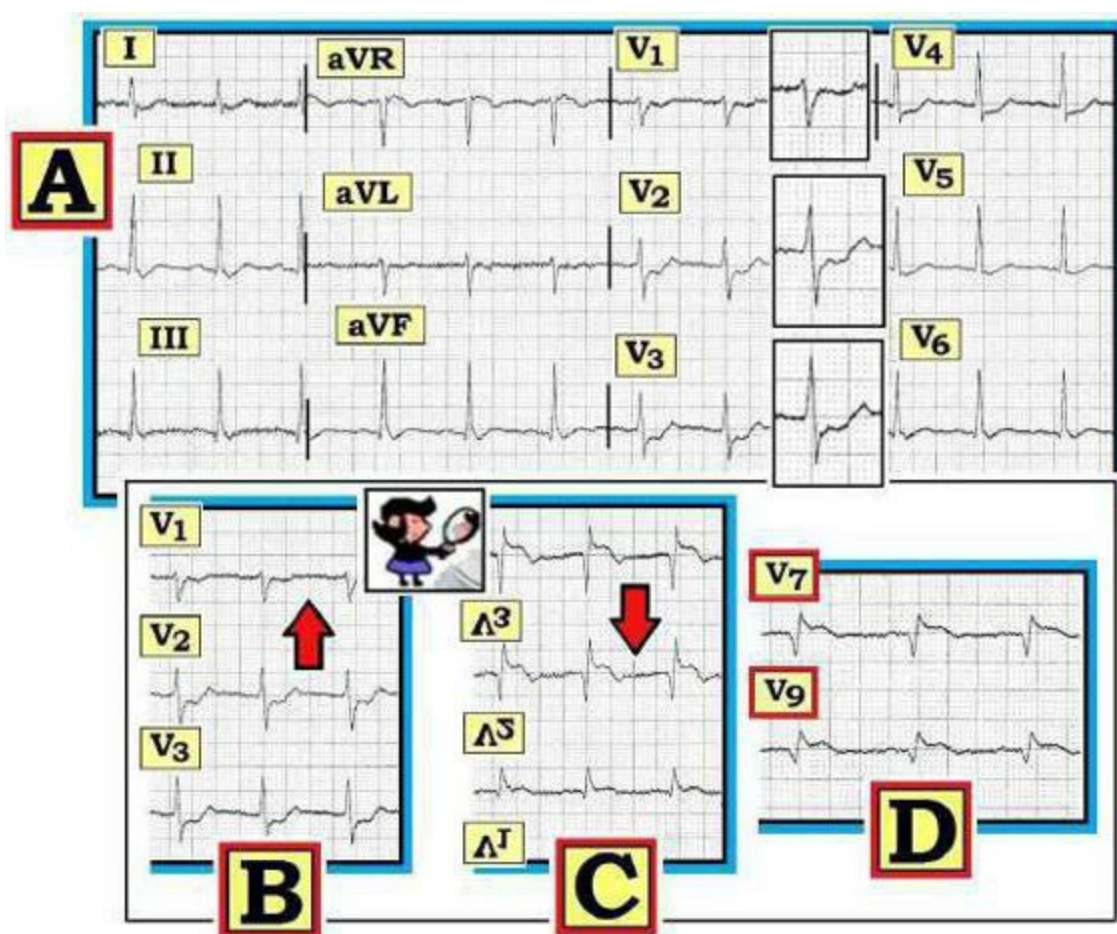


Figure 10.37-1: An example of *isolated posterior MI*. Diagnosis of acute *posterior* MI is suggested in **Panel A** by *anterior* ST depression (most marked in *V2, V3, V4*) and disproportionately tall R waves in *V2, V3*. A *positive “mirror test”* in **Panel C** (compared to upright leads *V1, V2, V3* in **Panel B**) supports this diagnosis. Use of *posterior* leads *V7* and *V9* showing Q waves and ST elevation confirms the diagnosis of *posterior* MI (**Panel D**).



PRACTICE: Acute MI

We consolidate principles covered thus far in discussion of the ECG diagnosis of *Acute MI/Ischemia* with a series of *Practice Tracings*.

- We begin with a full 12-lead tracing (*Section 10.39*).
- There follows a series of 12 *schematic* ECGs (*Section 10.40*).
- For *each* tracing — Identify ECG findings suggestive of ischemia/infarction. IF relevant — Try to identify the “*culprit*” artery in each case.

HINT: Feel free to refer back to previous segments on:

- ECG Changes of *Acute* Infarction (*Sections 10.1 through 10.10*).
- Sequence of ECG Changes (*Sections 10.11 through 10.15*).
- The Coronary Circulation (*Sections 10.16 through 10.21*).
- Identifying the “Culprit” Artery (*Sections 10.22 through 10.28*).
- Acute RV MI (*Sections 10.29 through 10.32*).
- Posterior MI/Mirror Test (*Sections 10 through 10.37*).

10.39 – FIGURE 10.39-1: *What is the “Culprit” Artery?*

Interpret the ECG in Figure 10.39-1 — obtained from a patient with *new-onset* chest pain.

- Can you localize the site(s) of *acute* infarction?
- What is the “*culprit*” artery likely to be?

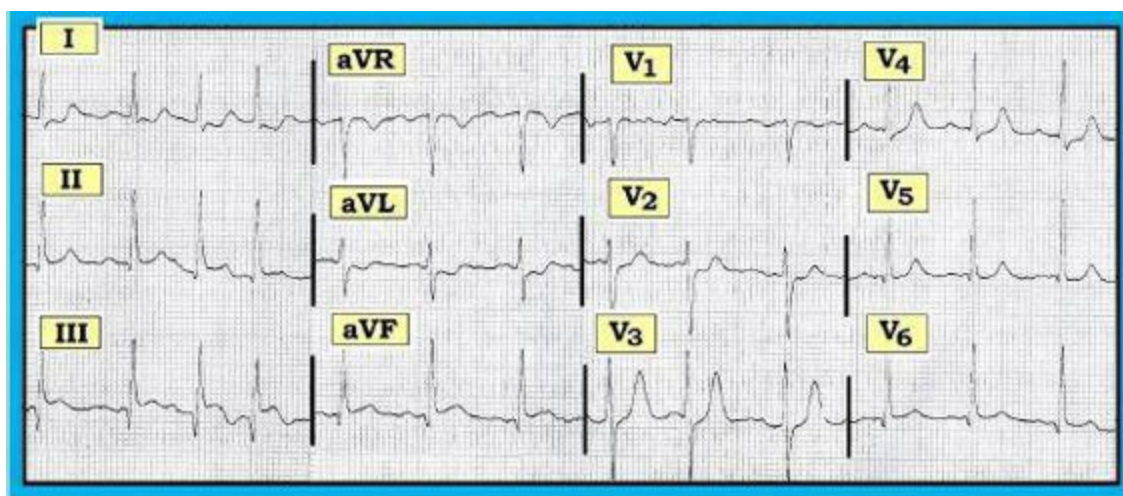


Figure 10.39-1: Sinus rhythm with PACs. Can you localize the site(s) of *acute* infarction? What is the “*culprit*” artery likely to be? (*See text*).

Answer to Figure 10.39-1: Although at first glance, the rhythm irregularity apparent for the first few beats looks like atrial fibrillation (*AFib*) — the rhythm then becomes more regular (*right after the first lead switch to leads aVR, aVL, aVF*). From this point on — **sinus P waves** with a constant PR interval precede QRS complexes in most leads. Thus, the **rhythm** is **sinus with PACs**. The QRS is of normal duration, and the QT interval is not prolonged. The axis is normal (*about +60 degrees*). There is no chamber enlargement. The most remarkable findings relate to QRST changes:

- **Q-R-S-T Changes:** There are **Q waves** in the **inferior leads (II, III, aVF)** — and *small* q waves are also seen in *lateral* precordial leads V5, V6. R wave progression reveals transition to be normal (*between V2-to-V3*) — albeit R wave amplitude in lead V3 appears to be somewhat *taller-than-expected* for this lead. The most striking findings relate to ST-T wave changes.
- There is **ST elevation** in *each* of the **inferior leads**. ST elevation is clearly *most* marked in **lead III**.
- There is **reciprocal ST depression** in leads **I** and **aVL**. This ST depression is *more* prominent in lead aVL.
- There is also *subtle-but-real* J-point ST depression in leads V2, V3, V4. Finally — there is prominent **T wave peaking** in **lead V3**, and perhaps to a lesser extent in lead V4.

In view of the history of *new-onset* chest pain — the ECG in Figure 10.39-1 is virtually diagnostic of **acute infero-postero STEMI**.

- As to the “culprit” artery — We strongly suspect **acute RCA occlusion** because: **i)** there is acute *inferior* MI with ST elevation in lead III > lead II; **ii)** there is significant *reciprocal* ST depression in lead aVL that is *at least as marked* as in lead I; and **iii)** there is *no* ST elevation in *lateral* precordial leads V5, V6 — as there generally is when the “culprit” vessel is a *dominant* LCx (*Section 10.28*).
- Support that QRS morphology and the ST-T wave changes seen in the *anterior* leads of Figure 10.39-1 is suggestive of acute *posterior* involvement — is forthcoming from a **positive “mirror” test** (*seen in Panel B of Figure 10.39-2*).

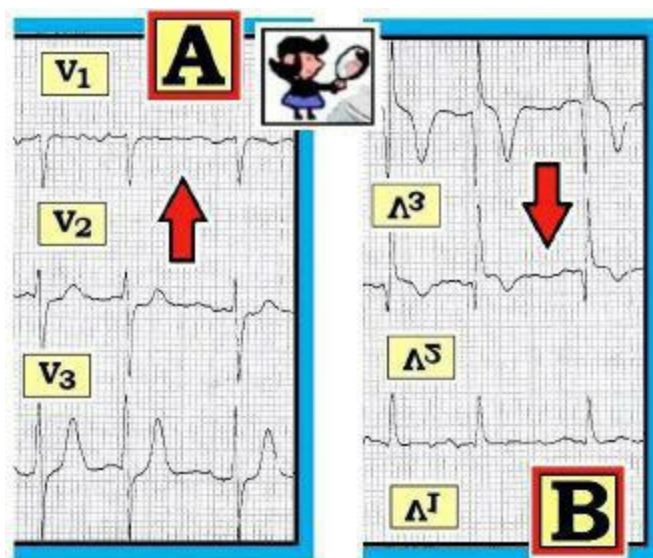


Figure 10.39-2: Leads V1,V2,V3 from the ECG in [Figure 10.39-1](#) are reproduced in **Panel A**. There is a **positive “mirror test”** in **Panel B** — in that the relatively tall R wave, depressed ST segment and *markedly* peaked T wave in lead V3 of [Panel A](#) — *becomes* a deep Q, elevated ST and deeply inverted T wave when the tracing is flipped over in **Panel B**.

Additional Points on Localizing the “Culprit” Artery: We expand on our rationale for strongly suspecting the RCA as the “culprit” artery for the ECG in [Figure 10.39-1](#). Practically speaking — the *majority* of **acute infero-postero STEMIs** will be the result of **RCA (rather than LCx) occlusion**, as ~85% of patients have a *right-dominant* circulation ([Section 10.17](#)). Recognizing that the *amount* of **ST elevation in lead III** is clearly *more* than in lead II — *strongly* favors RCA occlusion (*especially when there is significant reciprocal ST depression in lead aVL*).

- We next look to see IF there is **ST elevation** in **leads V5,V6**? IF there is — then the “culprit” artery could be either the RCA or a *dominant-left* Circumflex (LCx) artery (*since large postero-lateral artery branches may occasionally arise off the PDA from an RCA occlusion — as described in Section 10.18 — and as illustrated in Figure 10.28-3*).
- BUT — IF there is *acute infero-postero-lateral* STEMI but no ST elevation in leads V5,V6 (*as is the case in Figure 10.39-1*) — it becomes *highly likely* that the “culprit” artery is the RCA (*since one expects ST elevation in V5,V6 with left-dominant LCx occlusion*).

Beyond-the-Core: A final reason to support our suspicion of **acute RCA occlusion** for the ECG in [Figure 10.39-1](#) — is the ST-T wave appearance in **lead V1**, which shows a **flat ST segment** (*rather than ST segment depression*).

- In the presence of simple *acute infero-postero* MI — **lead V1** typically manifests a *similar-shape* ST segment as leads V2,V3. Thus, there should usually be *some* ST depression in lead V1 — albeit *less* than is usually seen in other anterior leads.
- IF instead of being depressed — the ST-T wave in lead V1 is flat or coved (or elevated) — then **associated acute RV (Right Ventricular) involvement** is likely. This is because the ST elevation from *acute* RV MI that we would *expect* to see in *right-sided* lead V1 — is in part *cancelled out* by ST depression from the *posterior* infarction ([Section 10.32](#)).
- IF there is a need to confirm acute RV involvement — then obtaining *right-sided* leads ([Section 10.31](#)) should tell us for certain.
- Identification of *acute* RV involvement localizes the “culprit” artery to the RCA — because the LCx does not supply the right ventricle.

10.40 – Schematic PRACTICE Tracings: Acute MI/Ischemia

As PRACTICE — We consolidate Review of the principles for *identifying* the probable “*culprit*” artery with *acute STEMI* in the following 12 *schematic* tracings (Sections 10.40.1 through 10.40.12).

- For *each* of these *schematic* ECGs — the rhythm is sinus and the patient presents with “*chest pain*”. Assess *each* tracing for evidence of *acute* infarction? If present — Can you identify the probable “*culprit*” artery?
- **NOTE:** To keep you honest — We include several *schematic* tracings that are not suggestive of *acute* STEMI. Can you recognize *what may be going on* in these cases?

HINT for the Tracings in Section 10.40: Feel free to refer back to previous segments on:

- ECG Changes of *Acute* Infarction (Sections 10.1 through 10.10).
- Sequence of ECG Changes (Sections 10.11 through 10.15).
- The Coronary Circulation (Sections 10.16 through 10.21).
- Identifying the “Culprit” Artery (Sections 10.22 through 10.28).
- Acute RV MI (Sections 10.29 through 10.32).
- Posterior MI/Mirror Test (Sections 10 through 10.37).

10.40.1 – FIGURE 10.40-1: Ischemia/Infarction?

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of *acute* infarction?
- If so — What is the “*culprit*” artery likely to be?

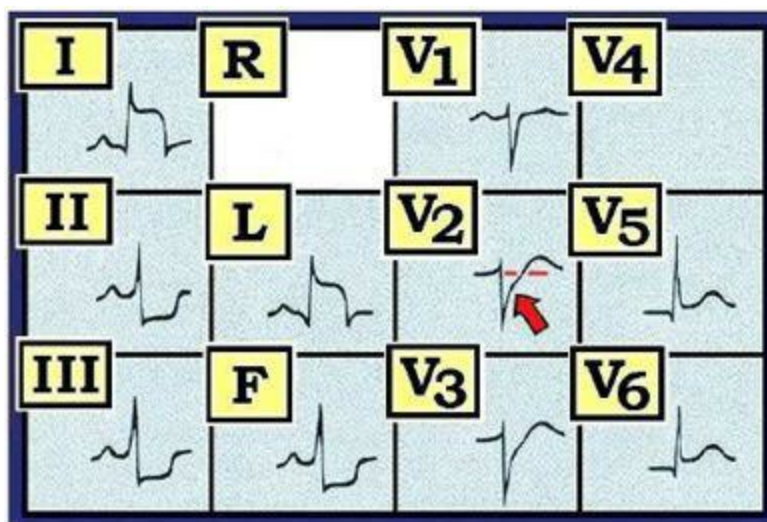


Figure 10.40-1: Schematic ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-1: There is *marked* ST elevation in *both* of the *high lateral* leads (*I,aVL*). In addition — leads I and aVL also manifest a *small* q wave and beginning T wave inversion. **Other *acute* findings** on this tracing include:

- **Leads V5,V6** — manifest a small q wave and ST elevation.
- **Inferior leads** (*II,III,aVF*) — manifest marked *reciprocal* ST depression.
- There is also *subtle-but-real reciprocal ST depression* in leads **V2,V3**. Note the *red arrow* in Figure 10.40-1 — which defines the **J-point** (*junction between the end of the S wave — and the beginning of the ST segment*). The slope changes at the J-point — with the ST segment seen to be significantly *below* the PR baseline.
- **Impression:** — Extensive **acute lateral STEMI** involving *high lateral* leads I,aVL and lateral precordial leads V5,V6.
- **Probable “Culprit” Artery:** — *Acute* occlusion of the LCx (*Section 10.28*).

Additional Comments on Figure 10.40-1: As emphasized in Section 10.28 — the lateral leads that most consistently assess the part of the heart supplied by the **LCx** are **leads V5,V6**. It will be rare indeed that you’ll encounter the combination of lateral lead findings shown in Figure 10.40-1, with near “*tombstone-type*” ST elevation in leads I and aVL. Thus, this *schematic* tracing is admittedly theoretical. Nevertheless, as a practice exercise — *lack* of inferior and anterior ST elevation suggests the LCx as the culprit artery.

- Finally — Note the small and **narrow q waves** in *lateral* leads I, aVL, V5 and V6 of Figure 10.40-1. Because these q waves are small and narrow — the question arises as to whether they might represent **normal septal q waves** *instead of* infarction q waves? Small septal q waves may normally be seen in healthy subjects in one or more of the lateral leads. That said — associated *dramatic* ST-T wave changes in this tracing strongly suggest they are **infarction q waves**. Subsequent tracings should clarify the issue — as we would expect these Q waves to deepen and widen as the infarct evolves.

10.40.2 – FIGURE 10.40-2: *Ischemia/Infarction?*

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

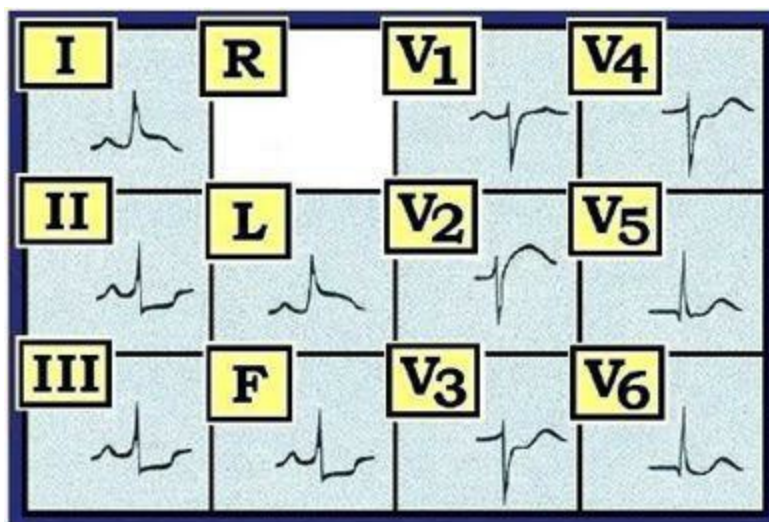


Figure 10.40-2: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “**culprit**” artery likely to be? (See text).

ANSWER to Figure 10.40-2: There is **ST elevation** in both **high lateral leads** (ie, in lead *I* and lead *aVL*). The only other lead showing ST elevation on this tracing is **lead V2**. This is indeed an unusual picture. **Other acute findings** include:

- **Reciprocal ST depression** — in the inferior leads (*II, III, aVF*) and in leads *V3, V4*.
- **Impression:** — Acute **high lateral STEMI**, with *in addition* ST elevation that is localized to lead *V2*.
- **Probable “Culprit” Artery:** — *Acute* occlusion of the **1st Diagonal branch** of the **LAD** (Section 10.25).

Additional Comments on Figure 10.40-2: The ECG picture shown in this schematic tracing is an uncommon but important pattern to be aware of. It emphasizes the **utility** of **ST elevation** in lead **aVL** for **localizing** the probable “**culprit**” artery. We repeat below the PEARL we presented in Section 10.25 on acute LAD occlusion:

PEARL: **ST elevation** in **lead aVL** — may provide an *invaluable* clue to the location of the acutely occluded coronary artery. According to a study by Birnbaum et al (*Am Heart J* 131:38, 1996):

- *Suspect* acute **LAD occlusion proximal** to the **1st Diagonal IF** *in addition* to ST elevation in *aVL* — there is *also* ST elevation in leads *V2*-through-*V5*. This is the most common situation when there is ST elevation in lead *aVL*.

- **Suspect 1st Diagonal branch occlusion** IF in addition to ST elevation in aVL — there is ST elevation in lead V2 (*but not in V3, V4, V5*).
- **Suspect LCx occlusion** (*especially of the 1st obtuse **marginal** branch*) — IF there is ST elevation in aVL but not in lead V2 (*and not in other anterior leads*).
- **NOTE:** Anterior ST elevation **without** ST elevation in lead aVL — suggests more *distal* LAD occlusion after takeoff of the 1st Diagonal.

Beyond-the-Core: We are aware of several cases in which awareness of the pattern shown in Figure 10.40-2 was of great assistance to the consulting cardiologist in identifying the site of acute coronary occlusion. A *totally* occluded 1st or 2nd LAD Diagonal branch may *not* always be readily apparent (*since no dye enters an occluded vessel*). Recognizing the specific *localizing* value of **ST elevation** in lead aVL and in lead V2 but not in any **other anterior leads** — helped the cardiologist to know exactly where to look on acute cardiac cath for the site of occlusion.

10.40.3 – FIGURE 10.40-3: Ischemia/Infarction?

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

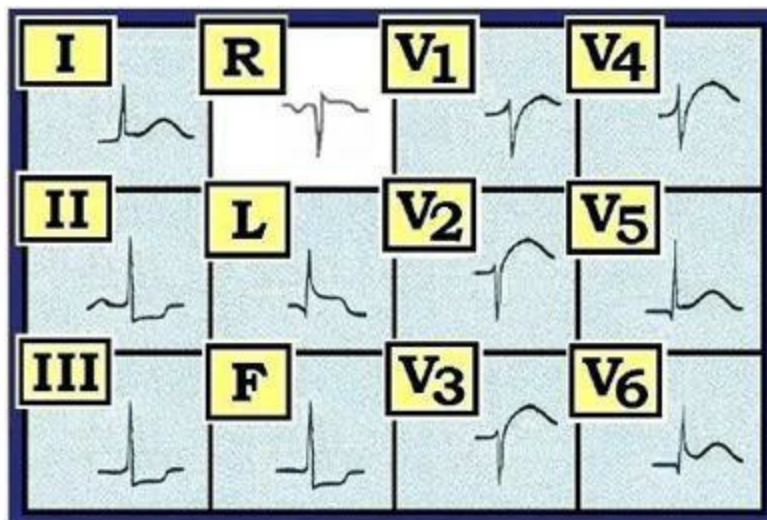


Figure 10.40-3: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-3: There is **ST segment coving** (“frowny” shape appearance) with **ST elevation** in the **precordial leads**. This is *most* marked in leads V2,V3,V4. **Other acute findings** on this tracing include:

- *Preservation* of the *initial* r wave in leads V1,V2,V3. This implies that the septum is still intact. That said — there is **loss of r wave** *between* lead V1-to-V2 (*in that* r wave amplitude decreases slightly from V1-to-V2). Normally, the R wave will be *taller-than-seen* in Figure 10.40-3 by lead V3.
- Several **other leads** manifest **ST elevation**. These include lead I, **lead aVL** and **lead aVR**.
- There is **reciprocal ST depression** in the *inferior* leads (II,III,aVF).
- Small and narrow q waves are seen in leads aVL,V5,V6.
- **Impression:** — Acute **extensive antero-lateral STEMI**.
- **Probable “Culprit” Artery:** — Acute occlusion of the **proximal LAD** (Section 10.25). As opposed to the case presented in Figure 10.40-2 — ST elevation in lead aVL is *accompanied by* diffuse precordial ST elevation. ST elevation is *also* present in lead aVR — but it is *not* markedly more than in lead V1. This localizes the likely site of acute occlusion to the *proximal* LAD.

Additional Comments on Figure 10.40-3: Note that the amount of ST elevation in lead V5 is not nearly as much as it is in neighboring leads V4 and V6. This highlights the concept of “**patterns of leads**”.

- Rather than looking at each of the 12 leads on a standard ECG as a separate entity — the ***experienced ECG “eye”*** *simultaneously* assesses acute changes in *specific* lead areas. Thus, *each* of the *inferior* leads (*II,III,aVF*) are best looked at together. In Figure 10.40-3 — leads II,III,aVF *all* show *similar* ST depression.
- Despite the relatively *minimal* amount of ST elevation in **lead V5** of Figure 10.40-3 — definite and marked ST elevation in ***neighboring leads*** (*V4 and V6*) tell us that *significant* ST elevation is truly present in *all* precordial leads!

10.40.4 – FIGURE 10.40-4: Ischemia/Infarction?

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

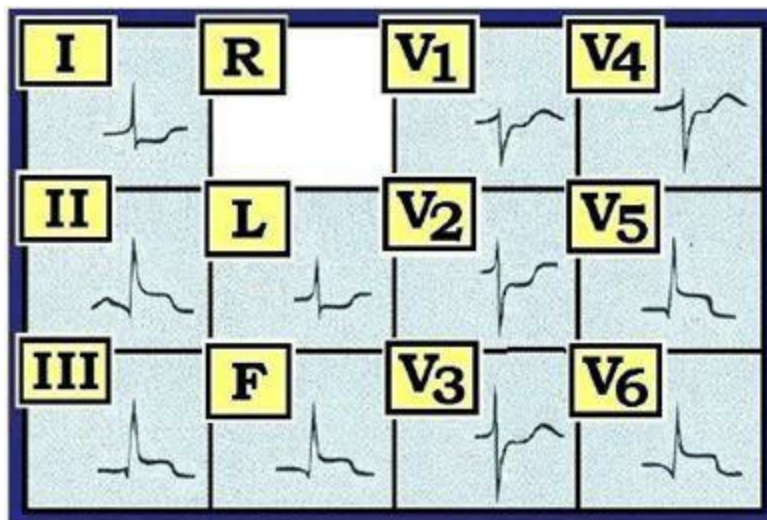


Figure 10.40-4: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-4: There are *both* **q waves** and **ST elevation** in the *inferior* and **lateral precordial leads** (*V5, V6*). Other **acute** findings on this tracing include:

- **ST depression** in leads I, aVL and in leads V1 *through* V4.
- There is a **positive “mirror” test** in the *anterior* precordial leads (Section 10.33).
- **Impression:** — Acute *infero-postero-lateral* STEMI.
- **Probable “Culprit” Artery:** — Acute occlusion of a **dominant LCx** (Section 10.28). This *schematic* tracing closely resembles the ECG picture previously seen in Figure 10.28-3. ECG findings on this tracing that strongly suggest the “**culprit**” artery is a **dominant LCx** (rather than the RCA) include: **i)** ST elevation in lead III is not more than in lead II; **ii)** ST depression in lead aVL is not more than in lead I; **iii)** There is **significant ST elevation** in leads **V5, V6** that appears to be more than the amount of ST elevation in lead III; and **iv)** There is *no* suggestion of acute RV involvement (ie, the ST segment in lead V1 is depressed and similar in shape to the ST depression seen in leads V2, V3).

Additional Comments on Figure 10.40-4: Note the presence of small and *narrow* **q waves** in *both* inferior and lateral precordial leads. The question arises as to whether these are likely to represent **normal septal q waves** vs *infarction* q waves?

- Small septal q waves may normally be seen in one or more of the *lateral* leads. While possible for small and narrow q waves to also be “septal” when seen in the *inferior* leads — this is *less*

likely (*especially when the patient does not have a vertical axis*). Given the overall ECG picture in Figure 10.40-4 — We strongly suspect that the *infero-lateral* q waves seen on this tracing are ***infarction* q waves** that will probably become *larger* as the infarct evolves.

10.40.5 – FIGURE 10.40-5: Ischemia/Infarction?

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

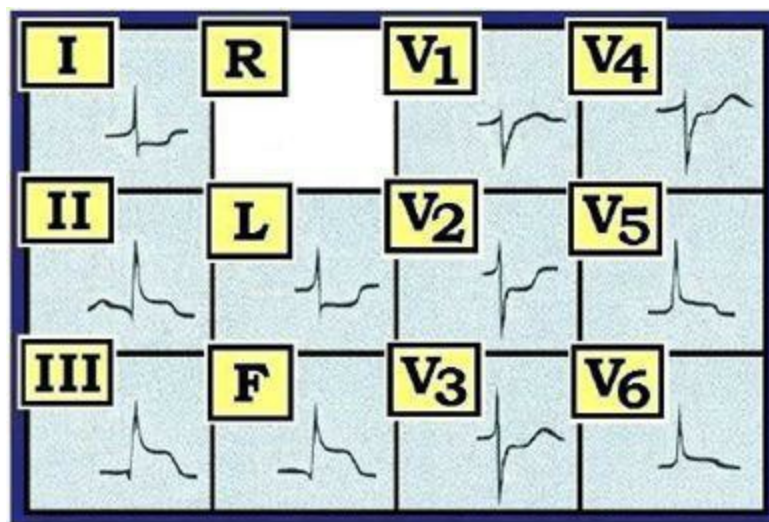


Figure 10.40-5: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-5: There is **ST elevation** in the *inferior* and *lateral precordial* leads (V5, V6). Other **acute** findings on this tracing include:

- Small q waves in the *inferior* leads.
- **ST depression** in leads I, aVL and in leads V1 through V4.
- There is a **positive “mirror” test** in the *anterior* precordial leads (Section 10.33).
- **Impression:** — Acute *infero-postero-lateral* STEMI.
- **Probable “Culprit” Artery:** — Acute occlusion of the *proximal RCA* (Section 10.23). ECG findings on this tracing that strongly suggest the “*culprit*” artery is the *proximal RCA* (rather than a dominant LCx) include: **i)** ST elevation in **lead III** that is clearly more than in lead II; **ii)** Marked ST depression in lead aVL that is *more* than in lead I; **iii)** No more than *minimal* ST elevation in leads V5, V6 — that is *less* in amount than the ST elevation seen in lead III; and **iv)** Suggestion of **acute RV involvement** by the upright T wave and level ST segment in *right-sided* lead V1. One usually expects to see ST depression in **lead V1** (similar to ST depression in leads V2, V3) when there is simple *posterior* MI without associated RV involvement (Section 10.32).

Additional Comments on Figure 10.40-5: The ECG picture in this schematic tracing illustrates an instance in which there may be ST elevation in leads V5, V6 that is not the result of acute LCx occlusion.

- On occasion — the **PDA** may give rise to large **PLA** (*Postero-Lateral Artery*) **branches** that extend laterally to supply parts of the LV free (*lateral*) wall. This may account for ST elevation in leads V5,V6 in a patient with acute RCA occlusion (*Section 10.18*). As noted above — there are no less than 4 reasons why we strongly suspect that the “culprit” artery in this case is the RCA (*and not the LCx*).

10.40.6 – FIGURE 10.40-6: Ischemia/Infarction?

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

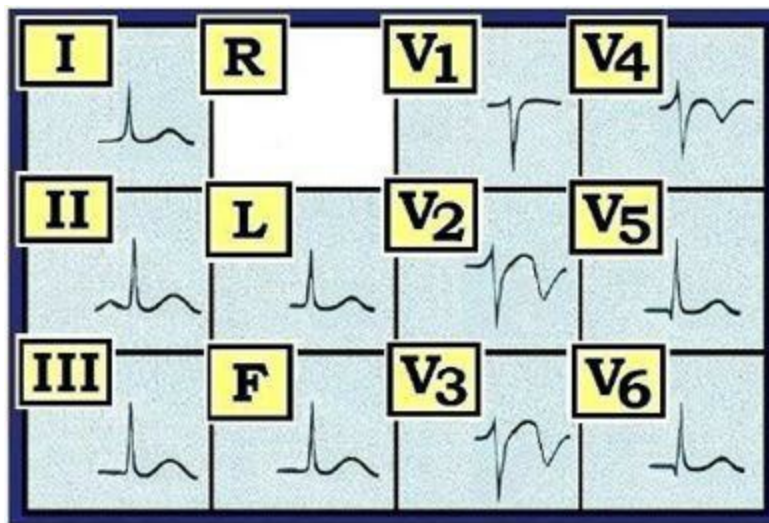


Figure 10.40-6: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-6: The most remarkable findings on this *schematic* tracing are seen in leads V2 through V4. Other than small, narrow (*septal*) q waves in leads V5,V6 — the rest of the tracing is unremarkable. We highlight the following:

- Although small — r waves are present in *each* of the anterior leads. Thus, there are *no* infarction q waves.
- The ST segment appears to be *coved* and *slightly* elevated in leads V2,V3. It is coved but *without* ST elevation in lead V4.
- There is a peculiar *very steep* downslope to the *initial* portion of the T wave in leads V2,V3.
- **Impression:** — This *schematic* tracing suggests **Wellens’ Syndrome**. As will be discussed in more detail in Section 10.54 — this ECG pattern is *highly* suggestive of a **critical stenosis** in the **proximal LAD**. The patient should be *immediately* referred for timely cardiac catheterization.

Additional Comments on Figure 10.40-6: Recognition of Wellens’ Syndrome is a *clinical* diagnosis. There should be a history of at least *some* symptoms consistent with coronary disease. These symptoms may be fairly acute — or they could be *ongoing* for a period of time. The ECG pattern of ST-T wave changes seen in Figure 10.40-6 may be intermittent (*due to slight changes in the degree of LAD obstruction*) — or it may become persistent.

- A *key* component to Wellens’ syndrome is that infarction has not yet occurred. Thus, although

there is *some* ST elevation in Figure 10.40-6 — it is *minimal* in amount and *no* Q waves have yet formed. This is important because the GOAL is to recognize Wellens' Syndrome before acute infarction (= *occlusion of the LAD*) occurs.

10.40.7 – FIGURE 10.40-7: *Ischemia/Infarction?*

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

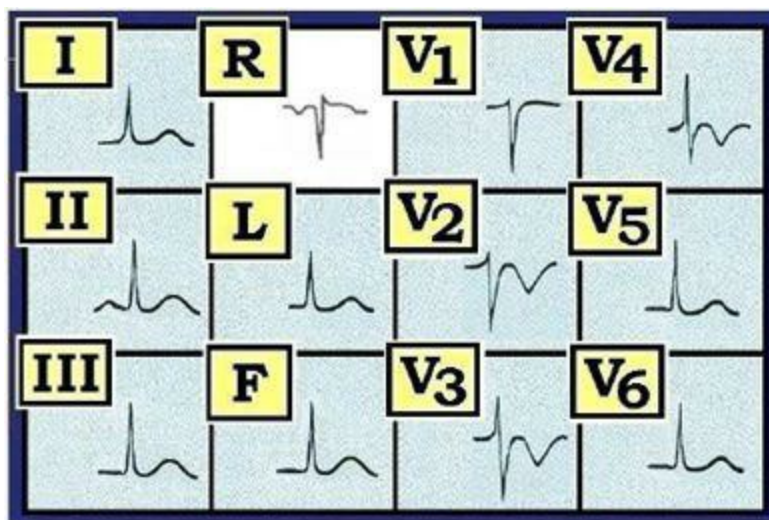


Figure 10.40-7: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “**culprit**” artery likely to be? (See text).

ANSWER to Figure 10.40-7: The remarkable finding on this *schematic* tracing is seen in the *anterior* leads — which show *fairly* deep and **symmetric T wave inversion** in leads **V2 through V4**. There are *no* Q waves — and R wave progression is normal (*transition occurs between lead V3-to-V4*). There may be *slight* ST elevation in lead aVR — but there are *no* other findings of note.

- **Impression:** — *Symmetric* T wave inversion in leads V2 through V4. This is consistent with **anterior ischemia**. *Strongly suggest clinical correlation!*

KEY Points: Clinical correlation is needed for *meaningful* interpretation of this *schematic* ECG. We simply can not tell from this *single* tracing IF the ECG finding of *symmetric* T wave inversion is new or old. **More history** and comparison with **prior** and **serial tracings** is needed.

- The findings in Figure 10.40-7 — could reflect recent *completed* infarction. Alternatively — these findings could reflect *ongoing* evolution of a *non-Q-wave* MI. Serial troponins may be needed to tell the difference.
- The *symmetric* T wave inversion seen here could reflect ischemia of *uncertain* duration. IF due to coronary disease — this could reflect significant narrowing of the LAD.
- Alternatively — T inversion could be due to some *other* cause (*including a noncardiac etiology*). For example — *anterior* T wave inversion may sometimes be an ECG indicator of *acute* pulmonary embolus (Section 08.37). **BOTTOM Line:** Clinical correlation is needed for *meaningful* clinical interpretation of this tracing.

10.40.8 – FIGURE 10.40-8: *How to “Date” an Infarct?*

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

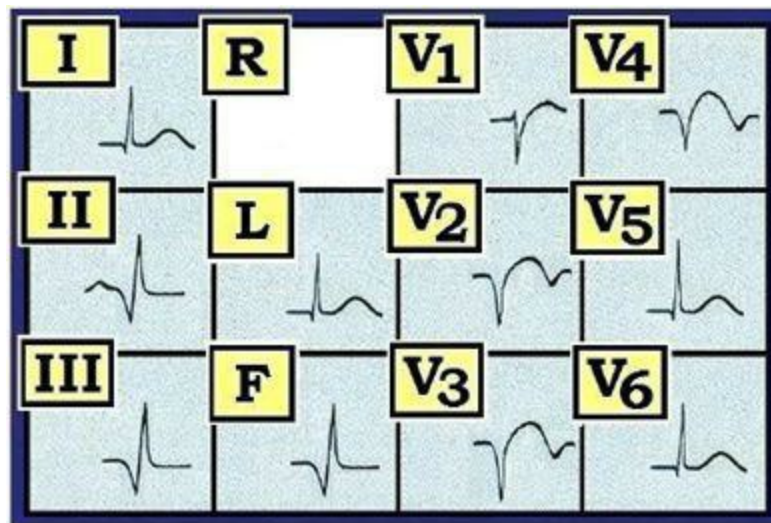


Figure 10.40-8: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-8: There are *many* important findings on this *schematic* tracing. These include:

- In the **inferior** leads — there are **large (and wide) Q waves** in leads II,III,aVF. These are associated with **flat ST segments**.
- In the **anterior** leads — there are **QS complexes** in leads V1 *through* V4. No R wave at all is seen until lead V5. **ST segments** in the **anterior** leads are **coved**, slightly **elevated** and associated with **T wave inversion**.
- **Impression:** — There clearly is evidence that infarction has taken place. The challenge is to attempt “**dating**” the one or more events that may have occurred (See below). That said — our interpretation of this *schematic* tracing would be as follows: i) **Old inferior MI**; and ii) **Anterior MI** of **uncertain** age, **possibly acute**. We would once again emphasize in our interpretation that, “*Clinical correlation is needed*”.

KEY Concept: How to “Date” an Infarct? When assessing a *symptomatic* patient for the possibility of *acute* ischemia or infarction — ECG terminology frequently refers to the presence or absence of “**acute**” changes. Given the importance of *prompt* revascularization for *acute* coronary occlusion — the GOAL is to identify *high-risk* patients with greatest potential for benefit. That said — Our ability to “**date**” an infarct is limited. Practically speaking — the *best* we can do is to classify ECG signs of infarction as *likely* to be: i) **Acute**; ii) **Old**; — or iii) Infarction of **Uncertain Age**.

- Examples of “**acute**” ECG findings are obvious in Figures 10.40-1 through 10.40-5. None of these tracings should pose any difficulty for recognizing the presence of *acute* MI.
- The *addition* of **History** to the ECG picture — may provide *invaluable* assistance for determining *onset* of the event. This is especially true when chest pain is severe and begins *abruptly*.
- In contrast — determining the likely “age” of an infarct *becomes* problematic when history for an “event” is indistinct (*minimal or only intermittent chest pain — or no chest pain at all*) — and when ECG signs are *less definite*. This is the case for assessing the **anterior leads** in Figure 10.40-8. The *very deep* QS complexes in leads V1 *through* V4 clearly suggest that **anterior MI** has occurred at *some* point in the past. Q waves of this depth (*QS complexes*) generally require a certain amount of time to develop. That said — ST segments in leads V2 *through* V4 of **Figure 10.40-8** are *coved*, slightly *elevated* and associated with T wave inversion. Whether this picture is due to recent *anterior* MI — *new* anterior MI (*superimposed on old infarction*) — or *persistent* ST elevation from ventricular aneurysm will require **clinical correlation** (*and comparison with prior tracings*) to sort out.

10.40.9 – FIGURE 10.40-9: Ischemia/Infarction?

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

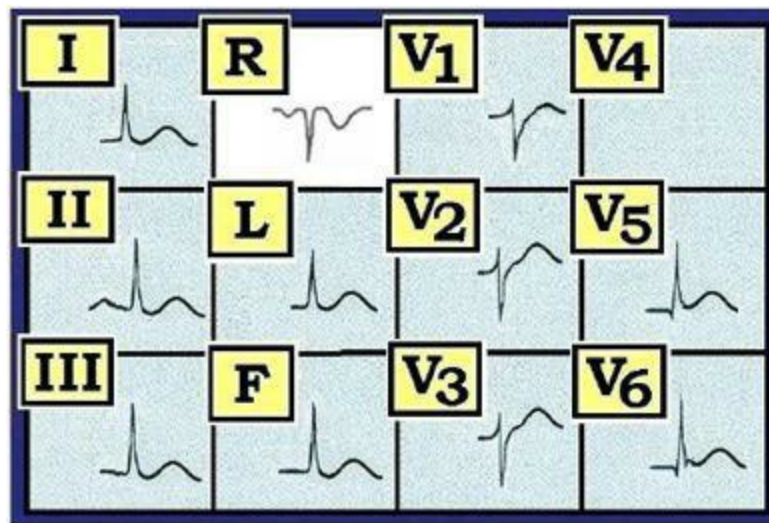


Figure 10.40-9: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-9: The ECG finding we wish to highlight on this schematic tracing is subtle: there is **slight ST elevation** in leads V2,V3 and in leads V5,V6. Given *smoothness* of the J-point in leads V2,V3 — it is difficult to be certain how much ST elevation is actually present. It does *not* appear to be much.

- The **shape** of **ST elevation** in leads V2,V3 is concave up (“*smiley*” configuration). It also manifests an *upward* concavity in leads V5,V6.
- There is **J-point notching** of the initial part of the ST segment in lead V6.
- Small and narrow q waves are seen in lateral precordial leads V5,V6. These look to be normal septal q waves.
- **Impression:** — Slight ST elevation in leads V2,V3 and V5,V6 with an *upward* concavity. J-point notching in lead V6. Small lateral q waves and *no* reciprocal ST depression. The overall pattern is most suggestive of **Early Repolarization** (Section 09.19). Strongly suggest *clinical* correlation!

Additional Comments on Figure 10.40-9: Clinical correlation is *essential* for *meaningful* interpretation of this tracing:

- If the patient in question was an otherwise healthy young adult and the history of “chest pain” was *not* suggestive of acute coronary disease — then we would *strongly* favor **Early Repolarization** as our clinical interpretation. The only q waves present are small — the *shape*

of ST segment elevation looks benign (*concave-up*) — there is J-point notching — and there are *no* reciprocal changes. (*Sections 09.17 through 09.21*).

- On the *other* hand — We would be *less* comfortable calling the ECG pattern in Figure 10.40-9 “benign” IF the patient was older, had risk factors and presented with *new-onset* severe chest pain.
- **BOTTOM Line:** Sometimes — “*Ya just gotta be there*” in order to make a clinical determination of how best to proceed. Availability of a ***prior tracing*** for comparison may prove invaluable for assuring that ST elevation is not new. On occasion — a tincture of time (*including serial tracings/troponins and a period of time to observe the clinical course*) may be needed.

10.40.10 – FIGURE 10.40-10: *Ischemia/Infarction?*

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

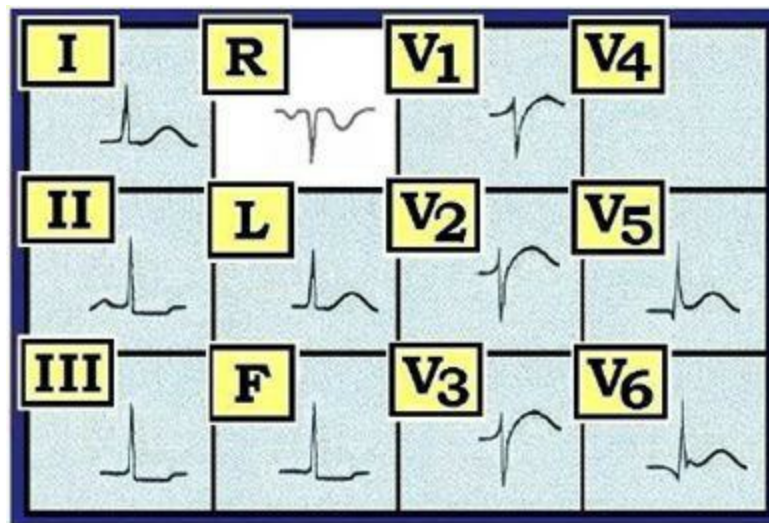


Figure 10.40-10: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-10: The *schematic* tracing shown here is similar in *some* regards to that from Figure 10.40-9 — in that there once again is **ST elevation** in **anterior precordial leads**. That said there are some *very* important differences between the 2 tracings:

- ST elevation is present not only in leads V2,V3 — but *also* in lead V1.
- The **shape** of **ST segments** in these leads is **coved** (“*frowny*” configuration). While possible that *coved* ST elevation could be a benign repolarization variant (See Section 09.24) — it is far more likely to indicate an acute coronary process (Section 09.17).
- There is *subtle-but-real* ST segment flattening and slight depression in *each* of the inferior leads (II,III,aVF). Given the findings seen in leads V1,V2,V3 — this qualifies as **reciprocal ST depression**.
- Small q waves with slight ST elevation is seen in leads V5,V6. There is J-point notching in lead V6. It is difficult to tell from this single tracing if this is a benign finding or something of possible concern.
- **Impression:** — We are suspicious of **acute anterior STEMI**. Lateral precordial ST elevation may or may not be part of this process.
- **Probable “Culprit” Artery:** — *Acute* occlusion of the **LAD** — though not necessarily in its proximal portion (Section 10.25).

10.40.11 – FIGURE 10.40-11: *Ischemia/Infarction?*

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

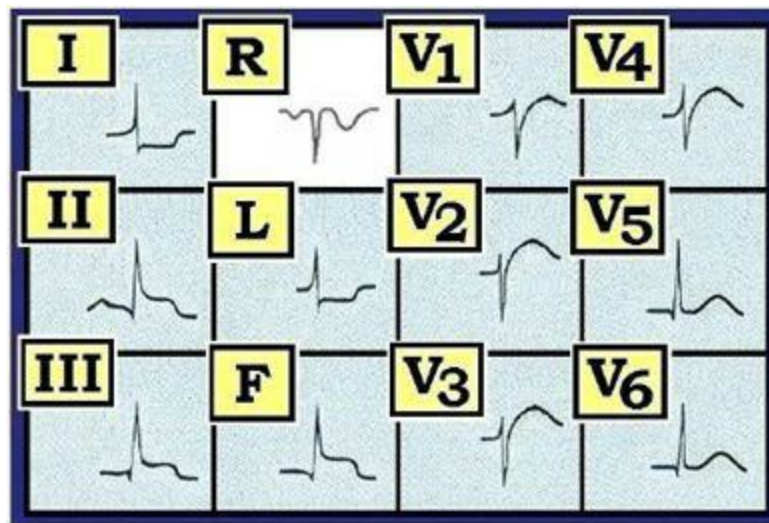


Figure 10.40-11: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-11: There are a number of findings of concern on this *schematic* tracing. These include:

- In the **inferior** leads — *small* Q waves with *definite* ST elevation.
- In the **anterior** leads — ST segment *coving* with *definite* ST elevation in leads V1 through V4.
- Small q waves are seen in leads V5, V6 — but there is no ST elevation in these leads.
- There is **reciprocal ST depression** in leads I and aVL.
- There appears to be *no* ST elevation in lead aVR.
- **Impression:** — The ECG picture clearly looks like acute *ongoing* infarction. In a patient with chest pain — we interpret these changes as suggestive of **acute inferior and anterior STEMI**. The question arises as to whether there is a *single* anatomic lesion that might give rise to these ECG changes that are seemingly occurring in 2 *different* lead areas?
- **Probable “Culprit” Artery:** — The left ventricular apex is often a difficult anatomic area to visualize on ECG. Changes may be subtle — or they may manifest in inferior *and/or* anterior leads. As emphasized in Section 10.27 — approximately 5-10% of normal subjects have an anatomic “**wrap-around**” LAD as a coronary artery variant circulation. In such cases — the LAD is a larger and longer vessel, to the point of extending *beyond* the cardiac apex and “*wrapping around*” to supply the undersurface (*inferior wall*) of the heart. Thus, we suspect *acute* occlusion of a “**wrap-around**” LAD as the “**culprit**” artery in this case (Section 10.27).
- As will be discussed in Section 10.61 — **Takotsubo Cardiomyopathy** is *another* potential cause of acute ST elevation in both inferior and anterior lead areas.

10.40.12 – FIGURE 10.40-12: *Ischemia/Infarction?*

The rhythm in this *schematic* tracing is sinus and the QRS complex is narrow. The patient is having “chest pain”.

- Is there ECG evidence of **acute infarction**?
- If so — What is the “**culprit**” artery likely to be?

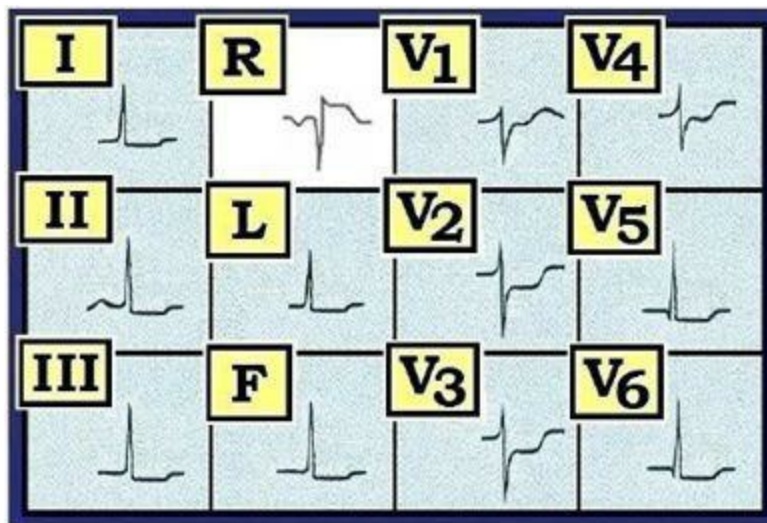


Figure 10.40-12: *Schematic* ECG from a patient with chest pain. Is there *acute* infarction? If so — what is the “*culprit*” artery likely to be? (See text).

ANSWER to Figure 10.40-12: The ECG picture in this *schematic* tracing is clearly of concern. There is marked **diffuse ST depression** in virtually *all* leads except for **ST elevation** in lead **aVR**.

- **Impression:** — The ECG pattern of *diffuse* ST depression in *multiple* leads (*usually in at least 7 leads*) — with ST elevation in lead aVR — is *highly* suggestive of **3-vessel** or **proximal LAD/left-main disease**. This is especially true when this pattern is seen in an *older* adult who presents with chest discomfort (*Section 09.40*).
- To emphasize *key* points regarding the recognition of this ECG pattern — We repeat below the PEARL and NOTE previously presented in Section 09.40:

PEARL: Distinction between *left-main* disease vs *proximal* LAD occlusion may be suggested on ECG by the **relative amount** of **ST elevation** seen in lead **aVR** *compared to* lead **V1**.

- Think **Left-Main** disease — when ST elevation in lead aVR > V1.
- Think **proximal LAD** disease/occlusion — when ST elevation in lead V1 > aVR. In Figure 10.40-12, given *marked* ST elevation in lead aVR in the *absence* of any ST elevation in lead V1 — one has to be concerned about significant *left-main* coronary artery narrowing.

NOTE: Distinction should be made between **acute LMCA** (Left Main Coronary Artery) **occlusion** vs **LMCA disease**.

- Most patients with *acute* LMCA occlusion do not survive. As a result — this entity is not often seen and *unlikely* to be appreciated clinically. Rapid deterioration with patient demise due to cardiogenic shock is the usual result unless acute LMCA occlusion can be *immediately* recognized and *immediately* acted on.
- In those *rare* circumstances when *acute* LMCA occlusion is captured on ECG — rather than diffuse ST depression there should be diffuse precordial ST *elevation* in association with significant ST elevation in lead aVR. Thus, the ECG picture in Figure 10.40-12 should not be misinterpreted as consistent with LMCA “occlusion”. Instead — it suggests that there may be significant LMCA *narrowing*.

10.41 – LIST #5: *Ant. ST Depression with Acute Inf. MI*



List #5: *Ant. ST Depression with Acute Inferior MI*

As discussed at the very *beginning* of this ECG-2014-ePub (*in Section 00.7*) — We have developed **6 Essential “Lists”** to remember for *optimal* ECG and arrhythmia interpretation. The purpose of a “List” — is that it readily recalls the most common/important entities to remember for the particular entity.

- We have already covered the first 4 of these Lists.
- For convenience — We **consolidate** all **6 Lists** in **Section 00.7** (*Make a bookmark — and/or Search for “00.7” to locate these 6 lists*).
- We present below in Section 10.42 our **LIST #5**.
- The 6th (*and last*) of our 6 Lists follows in Section 10.47.

10.42 – LIST #5: *Causes of Anterior ST Depression*

We have already reviewed a series of tracings showing *inferior* infarction. In the setting of **acute inferior MI** — **ST depression** is commonly seen in two or more **anterior leads** (*leads V1, V2 and/or V3*). This concept is illustrated in the *schematic* ECG shown below in **Figure 10.42-1**:

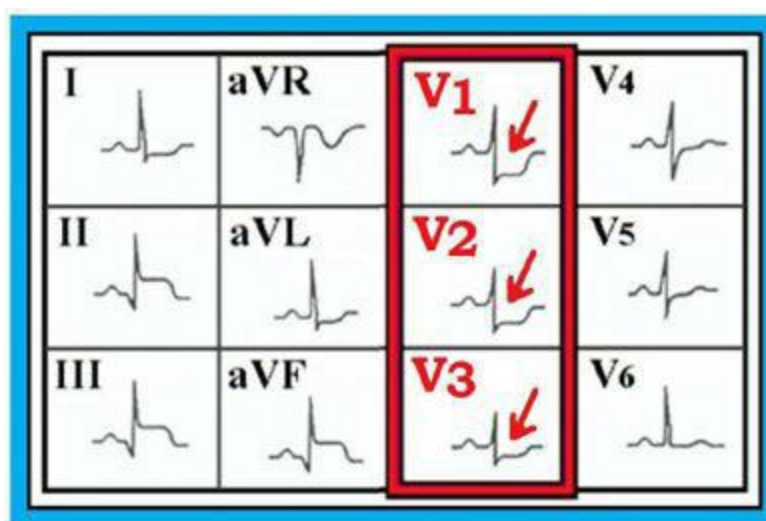


Figure 10.42-1: *Schematic ECG from a patient with acute inferior MI. Note the presence of anterior ST depression (in leads V1, V2, V3) — the common causes of which make up our LIST #5 (See text).*

LIST #5: The purpose of our 5th List is to facilitate recall of the 3 principal causes of **anterior ST depression** that commonly occur in the setting of **acute inferior MI**. *Each* of these 3 causes should be actively considered when confronted with an ECG such as that shown in *schematic Figure 10.42-1*.

- **CAUSE #1** — **reciprocal ST depression** (*that may occur in response to the acute inferior*

infarction).

- CAUSE #2 — **anterior ischemia**. Acute *inferior* MI is usually due to acute RCA occlusion. *Anterior* ischemia might be seen if *at the same time* there was symptomatic narrowing of the LAD.
- CAUSE #3 — **posterior MI**.

The above **3 Causes** make up our **LIST #5**. Keep in mind that *more than one* of these causes may be operative in any given patient (**Figure 10.42-2**):

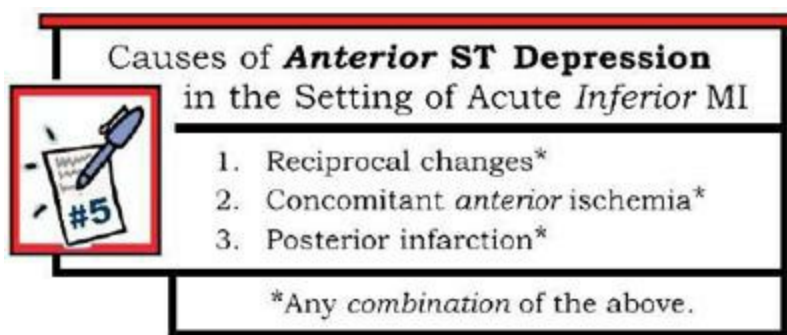


Figure 10.42-2: The *Common* Causes of *Anterior* ST Depression in the setting of Acute *Inferior* MI = **LIST #5**. More than one cause may be operative in any given patient (*See text*).

KEY Points about LIST #5: In any given patient — it may *not* be possible to distinguish which causes in List #5 are operative. That said — this does *not* matter clinically.

- *Regardless* of the cause(s) — **significant *anterior* ST depression** on ECG *in association with* acute *inferior* MI means a **larger infarct** (*and therefore more potential benefit from acute intervention*).
- **Acute *Posterior* MI** — will very often present when there is *anterior* ST depression in the setting of acute *inferior* MI. This is because the RCA most commonly supplies *both* the *inferior* and *posterior* wall of the LV (*Section 10.17*). Even when the “culprit” artery is a *dominant* LCx instead of the RCA — acute *inferior* and *posterior* infarction will commonly be seen together (*Section 10.17*). Use of the “*mirror*” test facilitates recognition of acute *posterior* MI (**Figure 10.42-3**).

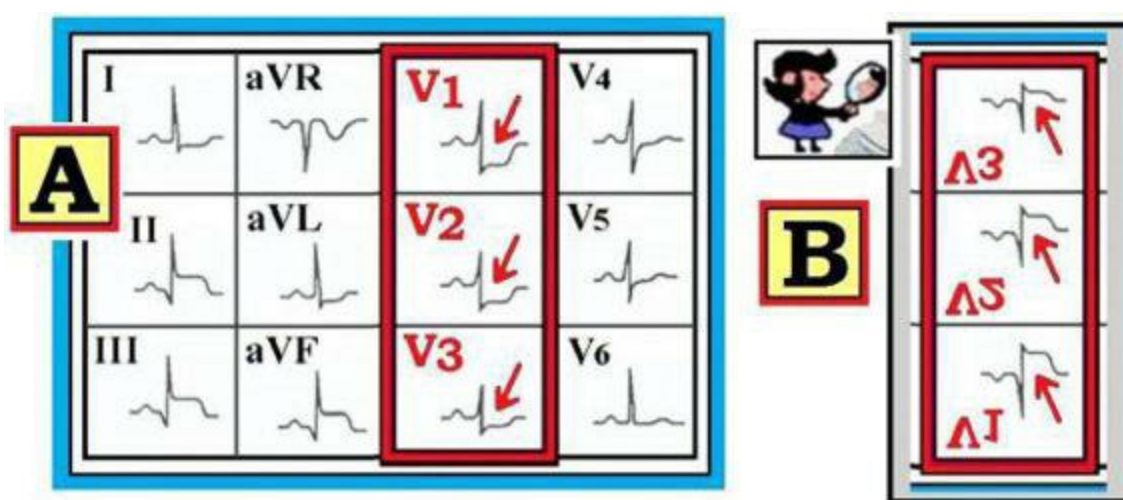


Figure 10.42-3: Application of the “*mirror*” test to Figure 10.42-1 (which we reproduce in Panel A). Flipping the tracing in **Panel A** over and holding it up to the light to perform the “*mirror*” test (**Panel B**) — supports the premise that **acute posterior MI** may be one of the causes of *anterior* ST depression that was seen in **Panel A** (See text).

10.43 – FIGURE 10.43-1: *Ant. ST Depression with Acute Inf. MI*

Practice applying List #5 by interpreting the ECG shown in **Figure 10.43-1** — obtained from a patient with *new-onset* chest pain.

- Is this patient having an *acute* STEMI? If so — What is the likely “*culprit*” artery?
- Note the **marked ST depression** in *anterior* leads **V1,V2,V3** (red arrows in Figure 10.43-1). What is the *likely* cause(s) of this ST depression?
- **HINT:** Feel free to refer back to **LIST #5** in Figure 10.42-2 in formulating your answer.

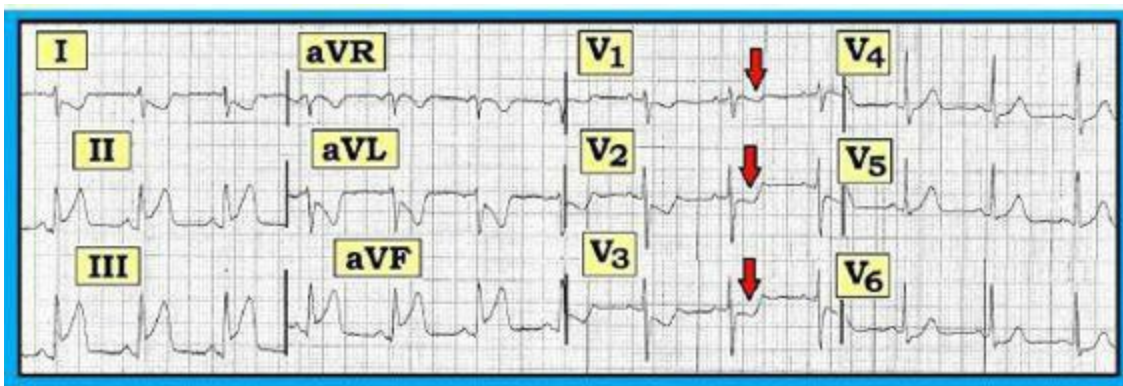


Figure 10.43-1: ECG from a patient with *new-onset* chest pain. What is the likely cause of the marked *anterior* ST depression? **HINT:** Feel free to refer back to **List #5** (in Figure 10.42-2) in formulating your answer.

Answer to Figure 10.43-1: The rhythm is sinus. This patient with *new-onset* chest pain is in process of evolving a large **acute infero-postero STEMI**. Relevant findings include the following:

- **Dramatic ST elevation** (with *hyperacute* ST segments) in *each* of the inferior leads. ST elevation in lead **III** is *more* than in lead **II** — suggesting the **RCA** is the “*culprit*” artery (Section 10.23).

- There is **reciprocal ST depression** in numerous other leads. This ST depression is especially marked in lead aVL — supporting the premise that there is acute RCA occlusion. ST depression is also seen in leads I; V1,V2,V3; and to a lesser degree in leads V4,V5,V6.
- Beyond-the-Core: Note that there is **marked RAD** (Right Axis Deviation) — as determined by the predominantly *negative* QRS complex in lead I. Given the *extensive* acute infarction seen here — this may reflect new *isolated* **LPHB** (Left Posterior HemiBlock — *discussed in Section 07.25*).

What is the Cause of Anterior ST Depression? The purpose of **List #5** — is to remind us of the 3 principal causes to consider when confronted with acute *inferior* MI and *associated* ST depression in the *anterior* leads (**red arrows in Figure 10.43-1**). These **3 Causes** are: **i) reciprocal** ST depression; **ii) anterior** ischemia from concomitant LAD narrowing; and **iii) posterior** MI.

- In **Figure 10.43-1** — We suspect at least 2 (*if not all 3*) of these causes are operative. ST depression is present in *multiple* leads in addition to leads V1,V2,V3. This suggests that *at least a component* of the *anterior* ST depression reflects **reciprocal changes**.
- Given the *extensive* size of the *acute* MI in **Figure 10.43-1** — there may well be **associated anterior ischemia**. This is especially true in view of the *marked* RAD (*probable* LPHB) — as the LAD more often provides blood supply to this hemifascicle.
- Finally — there *almost* certainly is associated **acute posterior MI**, which so often accompanies acute *inferior* MI (*especially when size of the infarct appears to be large*). Note *taller-than-expected* R wave amplitude in lead V2 of **Figure 10.43-1** — with the peculiar *shape* of ST depression in V2,V3 that characterizes acute *posterior* MI. The **“mirror” test** is *definitely* positive (**Figure 10.43-2**).

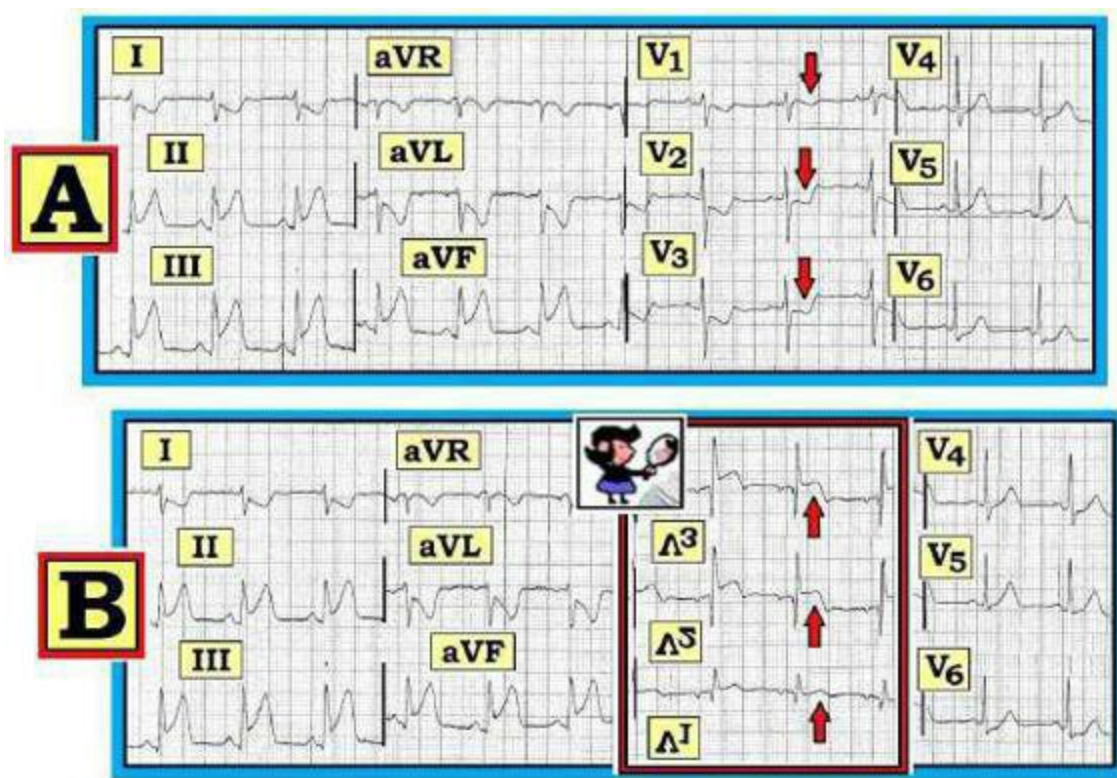


Figure 10.43-2: Note *anterior* ST depression (*red arrows*) in this patient with acute *inferior* MI (ECG in **Panel A** reproduced from Figure 10.43-1). Application of the “*mirror*” test in **Panel B** — which is *positive* and supports the premise that acute *posterior* MI is one of the causes of *anterior* ST depression that was seen in Panel A.

List #6: Tall R in Lead V1

As discussed at the very *beginning* of this ECG-2014-ePub (*in Section 00.7*) — We have developed **6 Essential “Lists”** to remember for *optimal* ECG and arrhythmia interpretation. The purpose of a “List” — is that it readily recalls the most common/important entities to remember for the particular entity.

- We have already covered the first 5 of these Lists.
- For convenience — We **consolidate** all **6 Lists** in **Section 00.7** (*Make a bookmark — and/or Search for “00.7” to locate these 6 lists*).
- We present below in Section 10.47 our *last* list = **LIST #6**.

10.45 – Normal Appearance of the QRS in Lead V1

As emphasized in Sections 05 and 09 — the **QRS complex** in **lead V1** will be **predominantly negative** under normal circumstances. This is because this *right-sided* lead (*V1*) normally sees electrical activity as *moving away* from V1 (*or toward the large left ventricle*). This concept is illustrated in *schematic* **Figure 10.45-1**.

- The finding of predominant *positive* activity in lead V1 (*an R wave that equals or exceeds the S wave in this right-sided lead*) — is not “normal”. This is the premise on which our List #6 is based (*Section 10.46*).

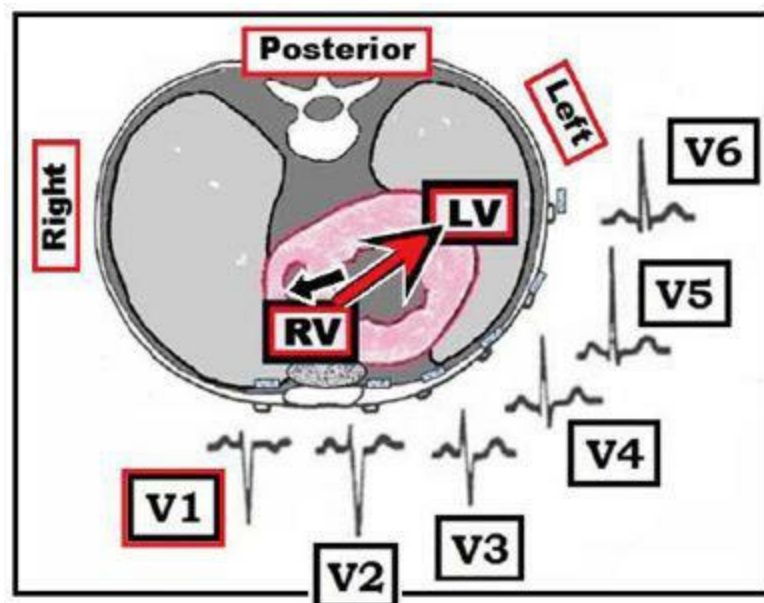


Figure 10.45-1: Transverse (*cross-sectional*) view of the heart — illustrating *precordial* lead appearance in leads V1-through-V6 (*reproduced from Figure 09.3-1*). Transition occurs in the above Figure *between* lead V2-to-V4. Note that the **QRS complex** in **lead V1** is **predominantly negative**

under normal circumstances (*red box*). Septal depolarization normally moves left-to-right (*small black arrow*). The major component of ventricular activation moves to the left and posteriorly (*large red arrow*) — which reflects the relative size and anatomic position of the left ventricle. This explains why lead V1 normally sees predominant electrical activity as moving *away* from this *right-sided* lead.

10.46 – The *Purpose* of List #6

It is *easy* to overlook the finding of a tall (*or relatively tall*) R wave in lead V1. It is equally easy to overlook the finding of *early* transition — in which the R wave in precordial leads V2 or V3 becomes *disproportionately* tall much *sooner* than expected.

- The *KEY* to *not* overlooking the ECG findings of a *tall* R wave in lead V1 or *early* transition — is to *routinely* apply a **systematic approach** to your ECG interpretation. This is our purpose for including the “**R**” *component* (*looking for **R** Wave progression*) when assessing for “**Q-R-S-T**” **Changes** (*Section 00.6.6*).
- The purpose of our **LIST #6** — is to facilitate recall of the principal causes of a *disproportionately* tall R wave in lead V1 (*Section 10.47*). Awareness of these causes is especially important — because *computerized* ECG interpretations typically *fail* to pick up a *taller-than-expected* R wave in leads V1,V2,V3.

10.47 – **LIST #6: Causes of a Tall R Wave in Lead V1**

In **LIST #6** — We note 6 important causes of a tall R wave in lead V1. The best way *not* to overlook any of these causes — is to work through *each* of the entities on this list (**Figure 10.47-1**) whenever you recognize that the R wave in lead V1 is taller than you expect.

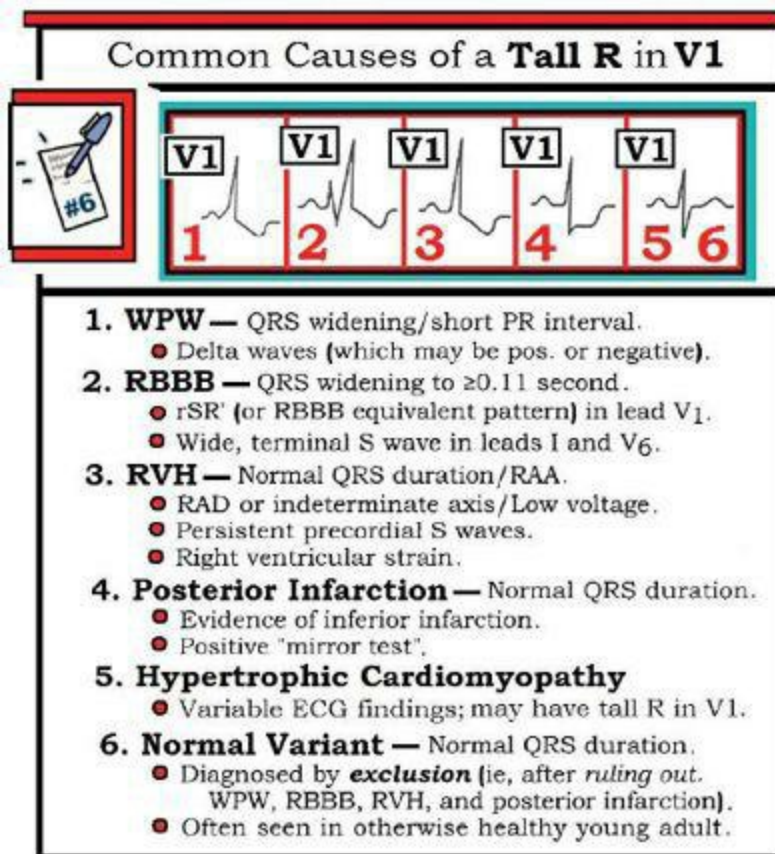


Figure 10.47-1: The Common Causes of a Tall R Wave in Lead V1 = **LIST #6**. Normal variant is a diagnosis of exclusion (See text).

Looking Closer at LIST #6: The way to *narrow down* which of the entities on List #6 is likely to be operative — is to look for *associated* findings.

- **WPW** — Look for the QRS to be wide with *delta* waves and a *short* PR interval (*discussed in detail in Section 05.37*).
- **RBBB** — Look for the QRS complex to be wide with an rSR' (*or equivalent*) in lead V1 and wide terminal S waves in leads I, V6 (*Section 05.4*).
- **RVH** — Look for ECG criteria of RVH including right or indeterminate axis; RAA (*Right Atrial Abnormality*); tall R wave in V1; RV “strain”; persistent precordial S waves (*Section 08.23*).
- **Posterior MI** — Look for ECG evidence of associated *inferior* infarction and for a *positive* “mirror test” (*Section 10.33*).
- **Cardiomyopathy** — increased *septal* forces from HCM (*Hypertrophic CardioMyopathy*) may manifest as a *disproportionately* tall R wave in lead V1 (*discussed in more detail in Section 10.49*). On occasion — *other* forms of cardiomyopathy (*such as that associated with muscular dystrophy*) may also result in a tall R wave in V1 (*Section 10.65*).
- **Normal Variant** — to be considered only after the above 5 causes have been *ruled out*. Thus, the diagnosis of “**normal variant**” as the reason for a disproportionately *tall* R wave in lead V1 — is a diagnosis of exclusion!

10.48 – PRACTICE Tracings: The Cause of the Tall R in V1?

As PRACTICE in *clinical* application of List #6 — We present the following 5 *schematic* tracings

and the real tracing in Figure 10.50-1:

- For *each* tracing — the rhythm is sinus and a **tall R wave** is seen in **lead V1**. Can you identify the *likely* cause of the *tall R wave* in lead V1 for *each* case? **HINT**: Feel free to refer back to **LIST #6** (in Figure 10.47-1) when formulating your answer.

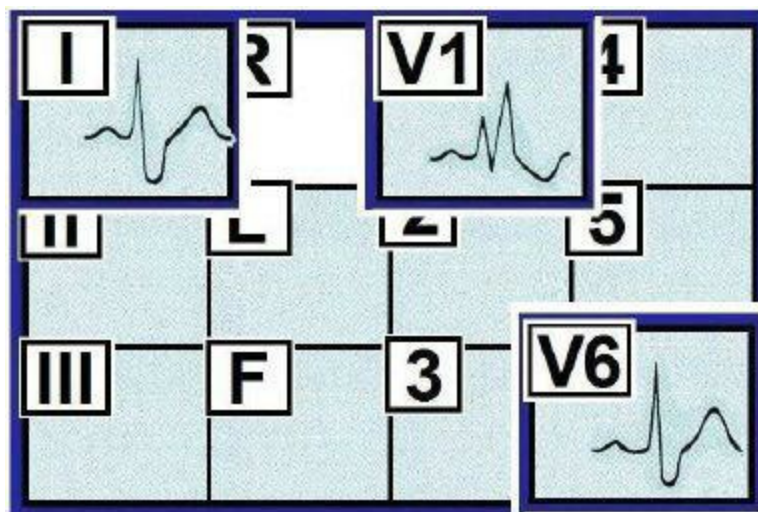


Figure 10.48-1: The rhythm is sinus. The QRS is wide. *Why the tall R wave in lead V1?*

Answer to Figure 10.48-1 The QRS is wide. A tall R wave with an RSR' (*taller right rabbit ear*) pattern is seen in lead V1. There are wide terminal S waves in leads I,V6. The cause of the *tall R* in V1 = **RBBB** (Section 05.4).

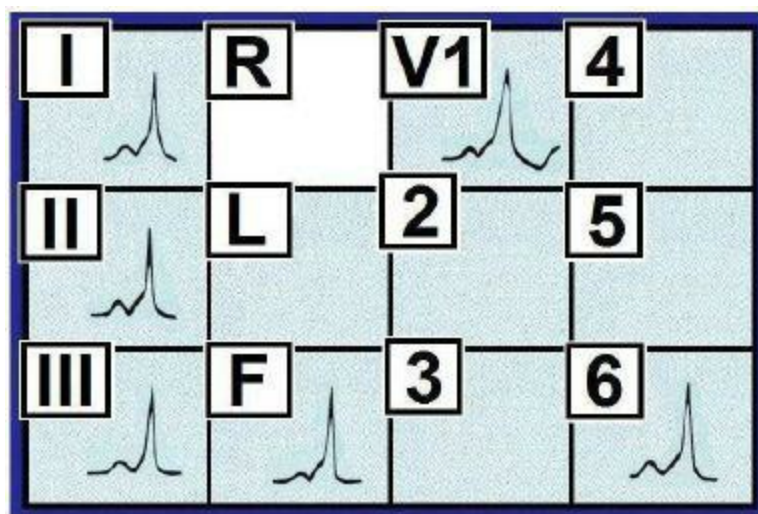


Figure 10.48-2: The rhythm is sinus. The QRS is wide. *Why the tall R wave in lead V1?*

Answer to Figure 10.48-2: The rhythm is sinus. The QRS is wide. The PR interval is *short* and there is initial *slurring* of the QRS upslope in the form of a **delta wave** in multiple leads. The cause of the *tall R* in V1 = **WPW** (Section 05.37).

- Note how this *schematic* example of WPW *simulates* RBBB in V1 — and *simulates* LBBB in leads I,V6. Remember — *delta waves* will not always be seen in all leads.

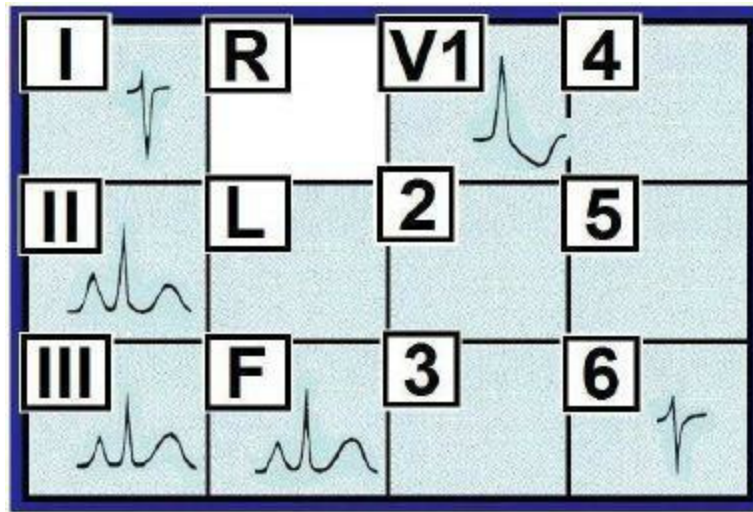


Figure 10.48-3: The rhythm is sinus. The QRS is narrow. *Why the tall R wave in lead V1?*

Answer to Figure 10.48-3: The rhythm is sinus and the QRS complex is narrow. The cause of the *tall* R in V1 = **RVH** (*Section 08.23*). Note the following features in support of this diagnosis:

- RAD (*Right Axis Deviation*) — as determined by the markedly *negative* QRS complex in lead I.
- RAA (*Right Atrial Abnormality*) — as determined by tall, peaked and pointed P waves in leads II,III,aVF.
- RV “strain” in lead V1 — as suggested by ST-T wave depression in V1 that occurs in association with the tall R wave in this lead.
- Persistent *precordial* S waves — as suggested by the deep S wave that is still present in lead V6.
- **KEY Point:** Remember to think of RVH as the “*detective*” **diagnosis** that is generally made by a *combination* of ECG findings that occur in the *right* clinical setting (*Section 08.24*).
- Practically speaking — By the time one sees as many ECG indicators of RVH in an *adult* as are present in *schematic Figure 10.48-3* — the patient has either: **i) end-stage** pulmonary disease; and/or **ii) pulmonary hypertension**.
- **NOTE:** The finding of a relatively *tall* R wave in lead V1 is much *more* common in children — where it is not necessarily abnormal during the first few years of life (*Section 08.31*).

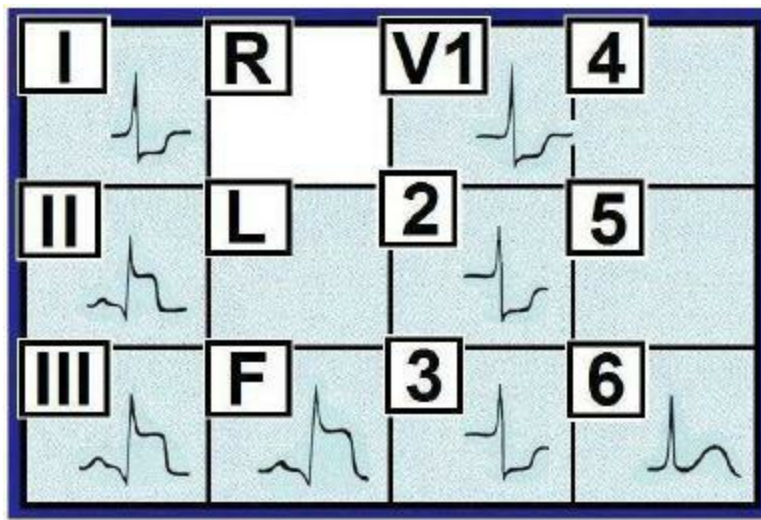


Figure 10.48-4: The rhythm is sinus. The QRS is narrow. *Why the tall R wave in lead V1?*

Answer to Figure 10.48-4: The rhythm is sinus. The QRS complex is narrow. There are small *inferior* Q waves and *marked* ST elevation consistent with **acute inferior MI**. ST depression is seen in leads I and in *anterior* leads V1,V2,V3. There is a **positive “mirror test”** (See below in **Figure 10.48-5**).

- **Impression:** We suspect the *tall* R in V1 is due to **acute posterior MI**. That said — the *other* 2 causes of *anterior* ST depression in LIST #5 may also be contributing (**Figure 10.42-2**). As emphasized in **LIST #5** — ST depression in leads V1,V2,V3 that occurs in association with acute *inferior* MI may be due to: **i) posterior MI**; **ii) reciprocal ST depression (in response to the acute inferior infarction)**; and/or **iii) anterior ischemia**.

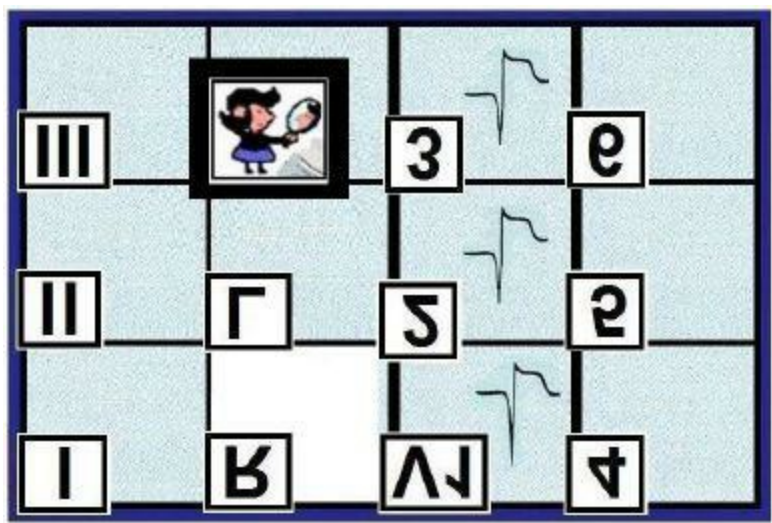


Figure 10.48-5: Application of the “mirror” test to the *schematic* tracing shown in Figure 10.48-4. The **postive “mirror” test** supports **acute posterior MI** as at least one of the causes of the *tall* R wave in lead V1 of Figure 10.48-4.

Finally — Consider the *schematic* tracing shown in **Figure 10.48-6**. The patient was an *asymptomatic* and otherwise healthy young adult. The ECG was obtained for screening purposes.

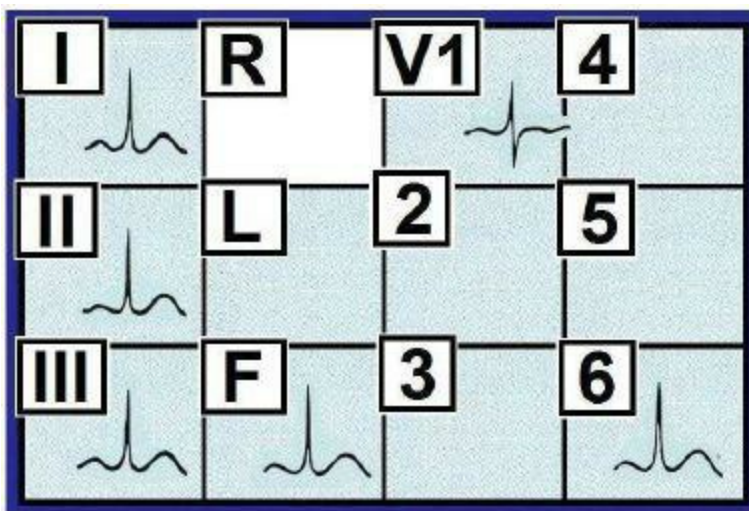


Figure 10.48-6: The rhythm is sinus. The QRS is narrow. The patient is an asymptomatic and otherwise healthy young adult. The ECG was obtained for screening purposes. *Why the tall R wave in lead V1?*

Answer to Figure 10.48-6: The rhythm is sinus. The QRS complex is narrow. We diagnose **normal variant** as the *likely* reason for the moderately *tall* R wave ($R=S$) in lead V1 — by the **process of elimination**. That is — the *other* causes in **List #6** must first be *ruled out*. We do this as follows:

- Since the QRS complex is *narrow* — the cause for the relatively *tall* R wave in lead V1 is not RBBB and not WPW.
- This is not posterior MI — because: **i)** the patient is an asymptomatic and healthy young adult; **ii)** there is no ECG evidence of inferior MI; and **iii)** the “mirror” test is not positive.
- This is not RVH — because: **i)** the axis is normal; **ii)** there is no RAA; **iii)** there is no RV “strain”; and **iv)** there is no persistent S wave in lead V6.
- Admittedly — We can not rule out **HCM** (*Hypertrophic CardioMyopathy*) without an Echocardiogram. This does not necessarily mean that one needs to obtain an Echo on all patients with an ECG such as seen in *schematic Tracing 10.48-6*. *Clinical judgement is needed*. IF this patient was an athlete competing in high intensity sports *and/or* had a heart murmur or *positive* family history of *early* sudden death — then an Echo is clearly indicated. It is not necessarily needed without any of these factors.

10.49 – Hypertrophic Cardiomyopathy: How to Recognize on ECG?

Be aware of the 5th cause in **List #6** of a *Tall* R in Lead V1 — which is **HCM** (*Hypertrophic CardioMyopathy*). Although *not* overly common — HCM is an important potential cause of sudden death (*especially in young athletes*). **Echo is diagnostic!** On the other hand — **ECG findings** are highly variable. These may include a moderately *tall* R wave in lead V1 as shown in **Figure 10.48-6** — and which indicates prominent *septal* forces. It might also include deep septal Q waves; LVH by voltage; IVCD/ LBBB — or *no* ECG changes at all. The reason for emphasizing *awareness* of HCM is the risk of *sudden death* that HCM poses among previously healthy young adults. While cost concerns prohibit mass screening by Echo of all young adults — Echo is indicated when there is a history of syncope during exercise; with a *positive* family history for *early* sudden death; when a *non-innocent* murmur is heard — or when a *pre-participation* ECG reveals *abnormal* findings that may

be consistent with the diagnosis.

10.50 – FIGURE 10.50-1: *WHY the Tall R in V1?*

We conclude this segment on recognizing the cause of a disproportionately *tall* R wave in lead V1 — with the ECG shown in **Figure 10.50-1**. The patient is a middle-aged adult. No other clinical information is available. *What is the cause of the tall R wave in lead V1?*

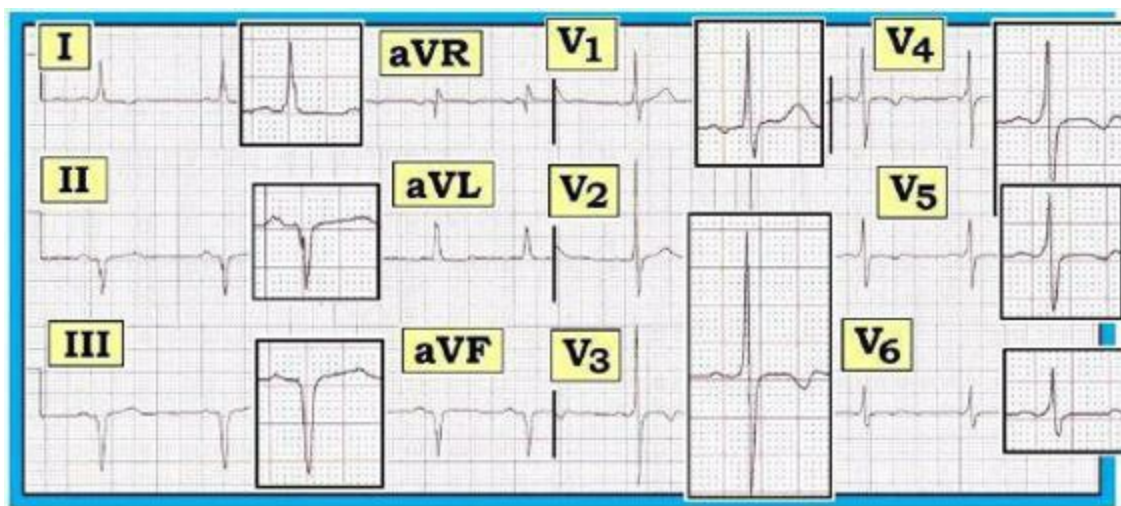


Figure 10.50-1: ECG obtained from a middle-aged adult. No other clinical history is available. *Why the tall R wave in lead V1?*

Answer to Figure 10.50-1: The QRS complex looks to be slightly wide. The rhythm appears to be sinus — as suggested by the presence of an upright P wave in lead II. The PR interval in lead II looks normal. The QT is not prolonged. The most remarkable finding on this tracing — is the **very tall R wave in lead V1**. This is clearly *not* expected — and should prompt consideration of the 6 entities in **LIST #6** as a possible explanation. We suspect that the answer will probably *also* explain: **i)** the marked left axis (*and/or QS complex in inferior leads*); **and ii)** ST flattening and shallow T inversion seen in multiple leads. As we work through the entities on **List #6** — We note the following:

- This is *not* a “normal variant” tracing. Other than the tall R wave in lead V1 — there is really nothing to suggest RVH (*no right axis; no RAA; no RV “strain” in lead V1*). And although it almost looks as if there are inferior Q waves — this is not the usual picture of inferior infarction, and the “mirror test” is *not* suggestive of posterior infarction.
- Finally — the patient does *not* have RBBB. There is no rSR’ in lead V1 — and no S wave is seen in lead I. The QRS complex is also not as wide as is generally seen with bundle branch block.
- The patient has **WPW**! It is important to appreciate that the QRS complex is *not* always overly wide with WPW. This is because there may occasionally be *simultaneous* conduction down *both* normal and accessory pathway — which will result in only *partial* pre-excitation (*Section 05.38*). It is *because of* awareness of **LIST #6** — that one looks *extra hard* for **delta waves** whenever the finding of a tall R wave in V1 is seen. Close inspection reveals such delta waves are seen (*red and blue arrows in Figure 10.50-2*).

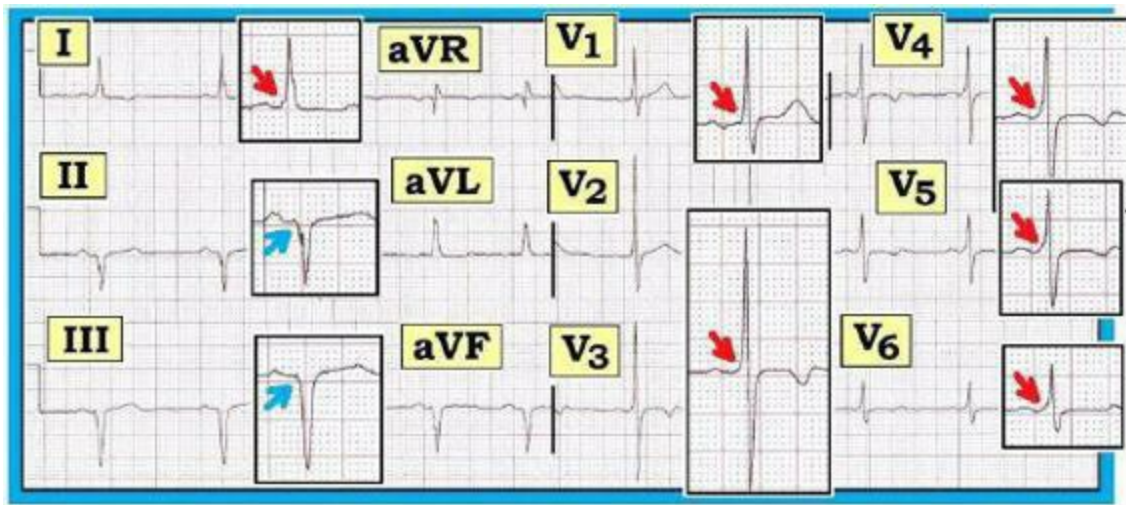


Figure 10.50-2: Arrows highlight delta waves that were subtly present in the ECG shown in [Figure 10.50-1](#). The QRS complex with WPW will *not* always be overly wide — as there may only be *partial* pre-excitation (if impulses are simultaneously conducted down normal and accessory pathway). Although the PR interval looks to be normal in lead II of this tracing — it appears to be *short* in leads V4, V5, V6 (red arrows in these leads). **Delta waves** are present. They are *negative* in the inferior leads (blue arrows) — and *positive* in other leads in which they are seen (red arrows). No delta wave is evident in leads aVR, aVL or V2.

10.51 – Giant T Wave Syndrome



We have already discussed on a number of occasions ECG recognition and clinical implications of T wave inversion. When **T waves** are *symmetrically inverted* (as they are in **Figure 10.51-1**) — the possibility of **ischemia** should be strongly considered.

- Depending on the history — ***other* entities** might also account for the *symmetric* T wave inversion seen in **Figure 10.51-1**. For example, this could be due to ***acute pulmonary embolus*** — IF the patient experienced *sudden* shortness of breath (*Section 08.37*). It could even be a ***normal variant*** (*Juvenile T wave pattern*) — IF the patient in question was an otherwise healthy and asymptomatic child (*Section 08.31*).

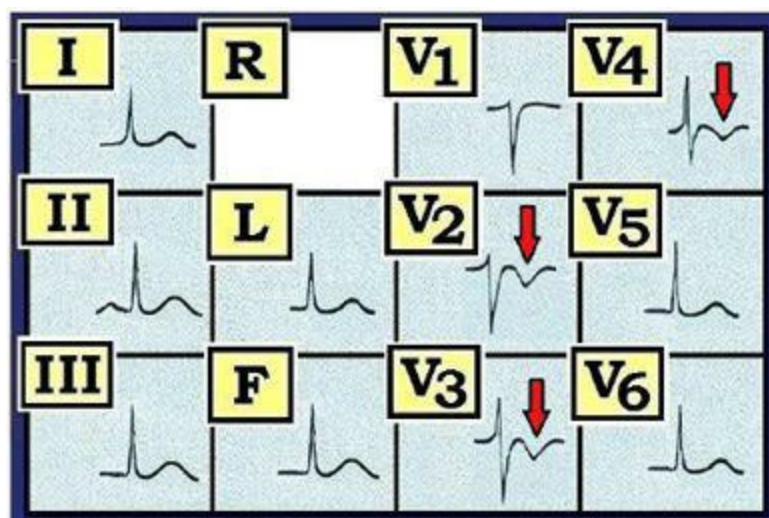


Figure 10.51-1: Schematic ECG illustrating ***symmetric* T wave inversion** in the *anterior* leads (red arrows in leads V2,V3,V4). This ECG picture should suggest ischemia — although depending on the history, *other* causes might also be considered (*See text*).

10.52 – When Inverted T Waves are GIANT in Size!

Although *some* T wave inversion is a common ECG finding — the occurrence of truly *huge* T waves that are inverted in *multiple* leads is much less often seen. We reserve the term, “***Giant T Wave Syndrome***” — for a *select* number of clinical entities that produce ***truly deep*** (>5mm amplitude) T wave inversion. An example of this phenomenon is shown in **Figure 10.52-1**:

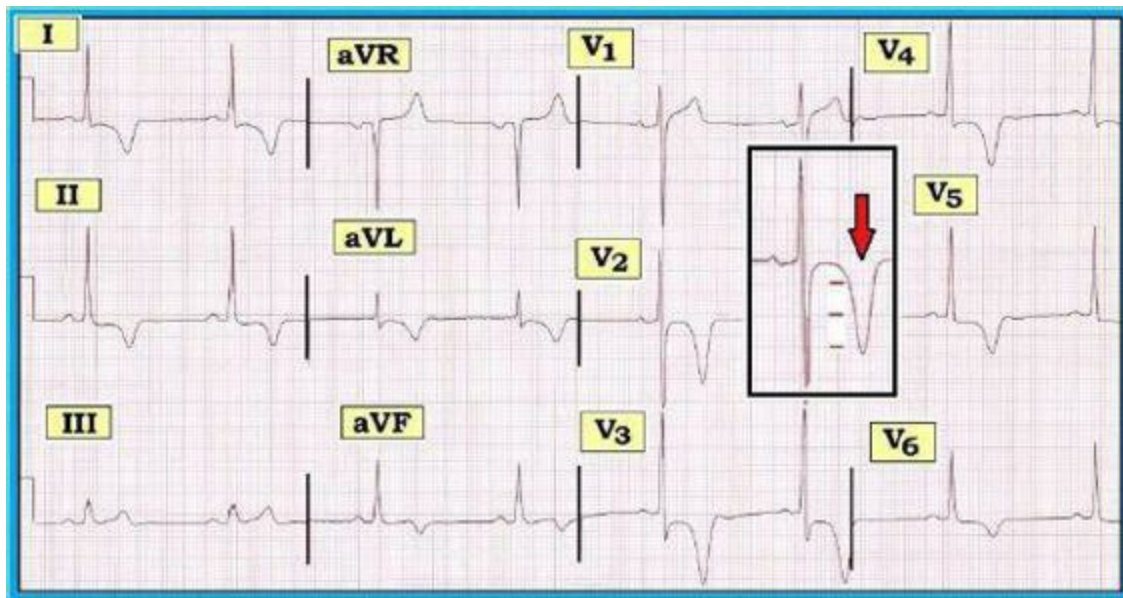


Figure 10.52-1: Sinus bradycardia with **Giant T Wave Inversion** that is present in *multiple* leads. As suggested by the *blow-up* insert — T waves *exceed* 10mm in size. A number of potential clinical causes should be considered (*See text*).

Causes of Giant T Wave Inversion: Think of the following **6 entities** when you see truly “*giant inverted T waves*” in *multiple* leads:

- Apical (*Yamaguchi*) Cardiomyopathy.
- Severe CNS disorders (*increased intracranial pressure*).
- Stokes-Adams attacks (*especially when due to severe bradycardia/complete AV block*).
- Anterior ischemia/coronary artery disease.
- Post-Tachycardia Syndrome.
- Massive Pulmonary Embolism (*acute right heart strain*).

Taking a Closer Look at the Causes: We highlight the following clinical points regarding the 6 common causes of *Giant T Waves*:

- *Giant T waves* were first described in association with **Stokes-Adams attacks** — in which patients presented with *syncope* from *complete AV block/bradycardia*.
- The most *bizarre* ECG changes occur with **severe CNS disorders** (*stroke, subarachnoid or intracranial hemorrhage, seizure, coma, brain tumors, trauma*). Often — the QT interval will be prolonged (*sometimes markedly so*) with CNS catastrophes. In addition to *giant T waves* — there may be *marked ST elevation* that *mimics* acute MI.
- **Apical (*Yamaguchi*) Cardiomyopathy** — is a special form of HCM (*Hypertrophic CardioMyopathy*) in which the left ventricular apex is disproportionately involved. *Giant T waves* are highly characteristic. Although Echo is the diagnostic procedure of choice for detecting HCM (*Section 10.49*) — localized *apical* thickening may occasionally be *missed* by Echo (*and only picked up by MRI scanning*).
- Deep, symmetric **anterior T wave inversion** — may suggest **coronary ischemia** from LAD (*Left Anterior Descending*) narrowing/occlusion (*Section 10.40.7*). Usually *other* clues in history (*chest pain*) or the ECG will be present — but on occasion, the diagnosis (*and indication to*

- perform cardiac cath) will only be forthcoming from *incidental* recognition of *giant* T waves.
- **Post-Tachycardia Syndrome** (as its name implies) — follows an episode of *sustained* tachycardia. This is a *transient* phenomenon after SVT or VT that does *not* indicate infarction. Usually T waves are *not* overly deep.
 - Finally — *anterior* T inversion may suggest acute RV “strain” and be a sign of **acute pulmonary embolism** (Section 08.37). The history should suggest *acute* PE; there should be *other* ECG signs (*right axis*; *RAA*; *fast rate*; *tall R in VI*).

10.53 – FIGURE 10.53-1: Cause of the Giant T Waves?

Return to the ECG that was shown in Figure 10.52-1 (which we reproduce below in **Figure 10.53-1**). IF told that the patient is an older adult who was found unresponsive — Which causes of **Giant T Wave Inversion** should be most strongly considered?

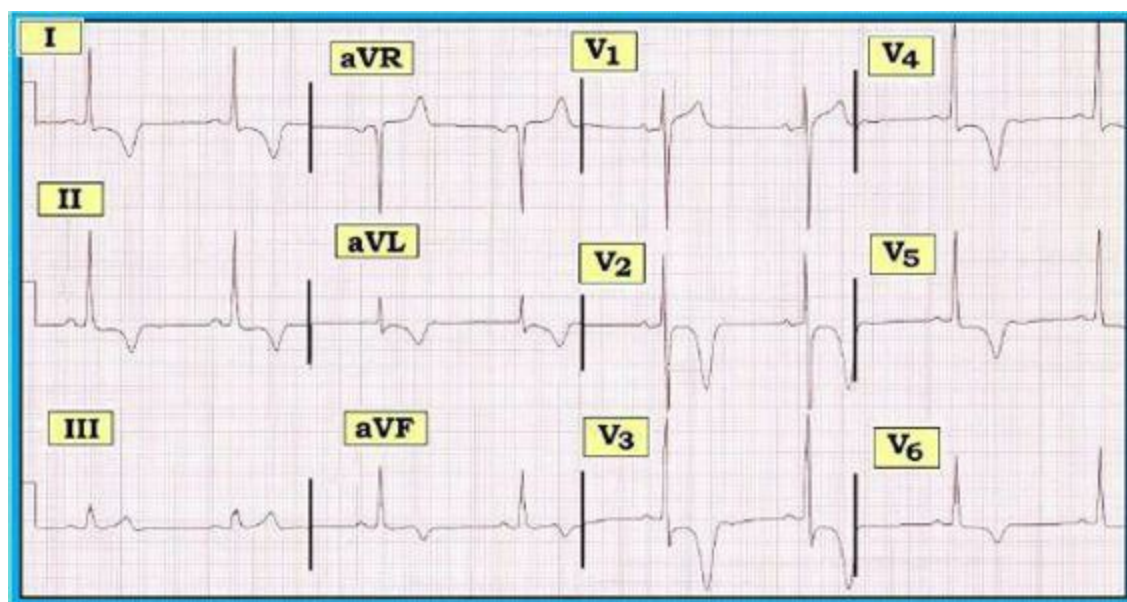


Figure 10.53-1: Sinus bradycardia with **Giant T Wave Inversion** that is present in *multiple* leads (reproduced from Figure 10.52-1). The patient is an older adult who was found unresponsive. What is the likely cause(s) of the giant inverted T waves? (See text).

Answer to Figure 10.53-1: The rhythm is sinus bradycardia. There is *marked* increase in QRS amplitude with *disproportionately* tall R waves in leads V1, V2, V3. Regarding ST-T wave changes:

- There is **Giant T Wave Inversion** — with *depth* of these inverted T waves easily *exceeding* 5mm in multiple leads.
- There is **ST segment coving** in several leads (V2, V3, V4) — albeit no significant ST segment elevation. There appears to be *slight J-point ST depression* in most of the leads that manifest T wave inversion.
- The **QT interval** appears to be *no more than slightly prolonged*. We measure the *actual* QT at over 0.50 milliseconds (*more than 2.5 large boxes in duration in lead V3*) — but given marked bradycardia, the QTc is really *not* overly long.

IMPRESSION: All of the entities discussed in **Section 10.52** should be considered as possible contributing causes of the *Giant T Wave Inversion* seen in **Figure 10.53-1:**

- This older adult has ***definite* LVH** (*marked increase in QRS amplitude; ST-T wave abnormalities consistent with “strain” and/or ischemia*). Echo (and possibly MRI) would be needed to determine IF the patient had ***Yamaguchi* (Apical) Cardiomyopathy**.
- Deep *symmetric* T inversion should always suggest **ischemia** — which may well be due to significant narrowing of the LAD. This would be more likely IF there was a history of chest pain prior to becoming unresponsive.
- Given this patient’s ***unresponsive state*** — the possibility of a **CNS catastrophe** (*stroke; bleed; trauma; post-seizure; metabolic disturbance*) needs to be strongly considered. That said — the QT interval will typically be *profoundly* prolonged when the cause of giant T waves is a CNS catastrophe, whereas the QTc in this case is *no more* than slightly prolonged.
- We have *no idea* if this patient’s unresponsive state *could have been* preceded by an episode of ventricular tachycardia or a Stokes-Adams attack resulting from profound bradycardia with AV block.
- Finally — *massive* pulmonary embolism may sometimes present with an unresponsive state, and could produce the giant T waves seen here.
- **BOTTOM Line:** All 6 entities listed in Section 10.52 should to be considered as possible cause of the *Giant T Waves* in **Figure 10.53-1**. Additional work-up will be needed to determine which cause(s) is likely to be operative in this case.

10.54 – Wellens' Syndrome



There are a number of instances in ECG interpretation — in which a particular ECG pattern correlates strongly with a *specific* anatomic entity. Perhaps the most important clinical examples of this phenomenon are: **i) Wellens' Syndrome**; and **ii) DeWinter T waves**:

- We address ECG recognition and clinical implications of **Wellens' Syndrome** below in Sections 10.55 and 10.56.
- Recognition of DeWinter T waves is addressed in Section 10.57.

10.55 – Wellens' Syndrome: Clinical Implications & ECG Recognition

In a small but *significant* percentage of patients who present with ischemic heart disease — the *initial* ECG may be *strongly* suggestive of a nearly (*but not totally*) occlusive pattern. **Prompt recognition** is essential in such cases — and may prove invaluable as a myocardial *sparing* and potential *life-saving* measure.

- Consider the ECG shown in **Figure 10.55-1** — obtained from a patient with *intermittent* chest discomfort. How would you interpret this ECG?
- What intervention is needed?
- What *anatomic* lesion is suggested?

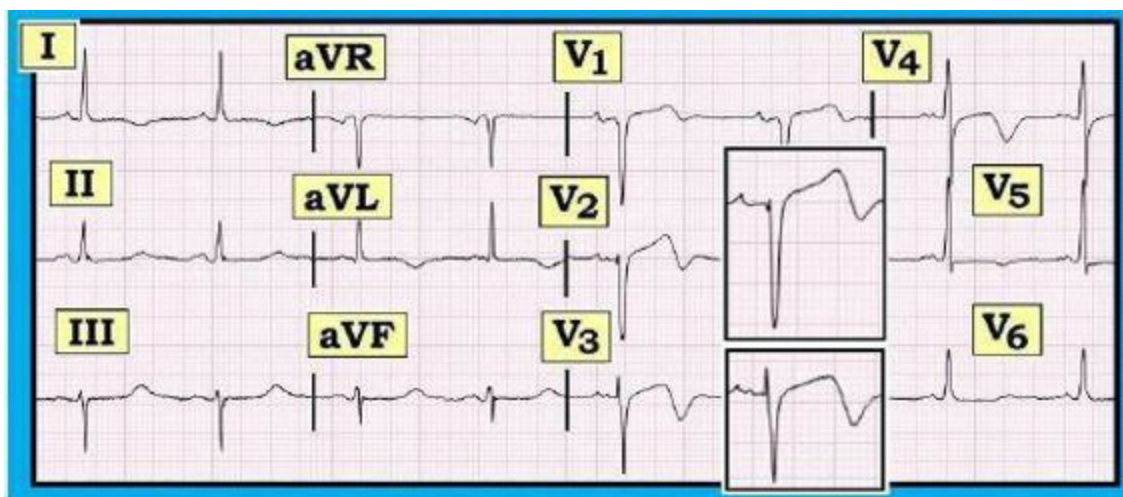


Figure 10.55-1: *Wellens' Syndrome*. This patient had *intermittent* chest pain. What intervention is needed? What *anatomic* lesion is suggested?

Answer to Figure 10.55-1: First introduced by Wellens and his group in 1982 — the ECG pattern seen in Figure 10.55-1 has been found to be **highly predictive** (*with ~90% accuracy*) of **critical narrowing** in the *proximal* LAD (*Left-Anterior Descending*).

- Identification of this ECG pattern (*known as Wellens' Syndrome*) — is indication for **prompt cardiac catheterization** — with expectation that *revascularization* will *likely* be needed.

We highlight the following **KEY Clinical** and **ECG Features** of **Wellens' Syndrome**:

- There is a history of *prior* angina or chest discomfort.
- There is little or no elevation of cardiac markers (*troponins*).
- *No* pathologic precordial Q waves.
- Slight (*but not marked*) ST elevation in *precordial* leads.
- Progressive and *symmetric* T wave inversion that may be diffuse (*especially in precordial leads*).
- A characteristic **abrupt takeoff** (*steep angle*) to the **deep T wave inversion** that is seen in **leads V2,V3 and/or V4**.

KEY Clinical Point: The importance of recognizing the above features of Wellens' Syndrome — is that there is a very **high incidence** among these *symptomatic* patients who have not yet infarcted (*no more than minimal troponin elevation; no significant Q waves; minimal ST elevation*) — of going on to develop **extensive infarction** IF they do *not* undergo prompt revascularization.

FIGURE 10.55-1: What Features are Seen in this Case? The ECG shown in Figure 10.55-1 *strongly* suggests **Wellens' Syndrome**. We note the following findings:

- There is a history of *intermittent* symptoms (*chest pain*).
- There is **slight** (*but not marked*) **ST elevation** in *anterior* leads V2,V3,V4 (*and perhaps also in lead V1*).
- The ST segment in these leads is straightened (*with a hint of downward coving in V3,V4*).
- Given this *slight* ST elevation in leads V2,V3 — the **downward slope** of the **T wave inversion** to follow in these leads is **remarkably steep**!
- There is *not yet* sign of definite infarction (*no Q wave in leads V2,V3,V4*).
- **Clinical Course:** This patient was found to have a >95% *proximal* LAD lesion. He was stented with excellent result (**Figure 10.55-2**).

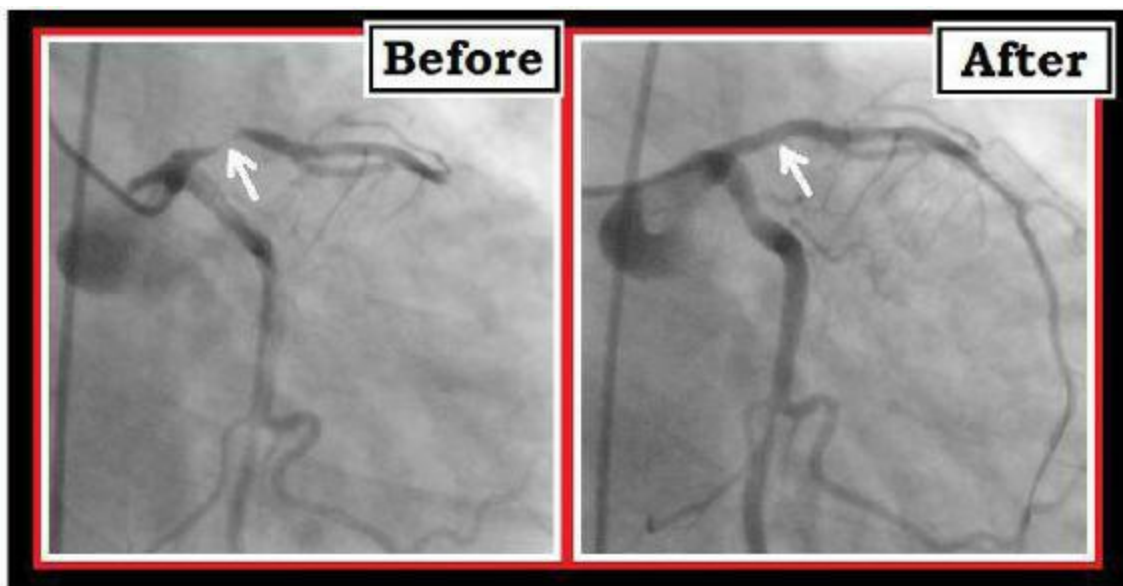


Figure 10.55-2: Cath films “Before” and “After” stenting the tight *proximal* LAD lesion from the patient whose Wellens’ Syndrome ECG was shown in [Figure 10.55-1](#). (Acknowledgement: My appreciation to Jason Roediger for contributing this case, including the ECG and cath films).

10.56 – FIGURE 10.56-1: *What Wellens’ Syndrome is Not!*

There are 2 ECG patterns that have been associated in the literature as representing “Wellens’ Syndrome”. We favor use of only one of these patterns to define the syndrome ([Figure 10.56-1](#)):

- Many clinicians interpret the pattern of **symmetric anterior T wave inversion** as one expression of *Wellens’ Syndrome* (**Panel A** in [Figure 10.56-1](#)). We feel this is a mistake that *defeats* the goal of recognizing this syndrome.
- While anterior *symmetric* T inversion (as seen in **Panel A** of [Figure 10.56-1](#)) clearly *does* occur in patients with ischemic heart disease — seeing this pattern on ECG without slight ST elevation and the **steep T wave downslope** in **Panel B** is simply not nearly as predictive of a proximal *critical* LAD lesion. Instead — patients who only manifest the *symmetric* T inversion of **Panel A** might have: **i)** milder (*non-critical*) coronary disease, which could be in *any* part of the LAD (*not necessarily the proximal LAD*); **ii)** any of the *other* causes of T wave inversion that were listed in Section 10.52; or **iii)** *no* heart disease at all.

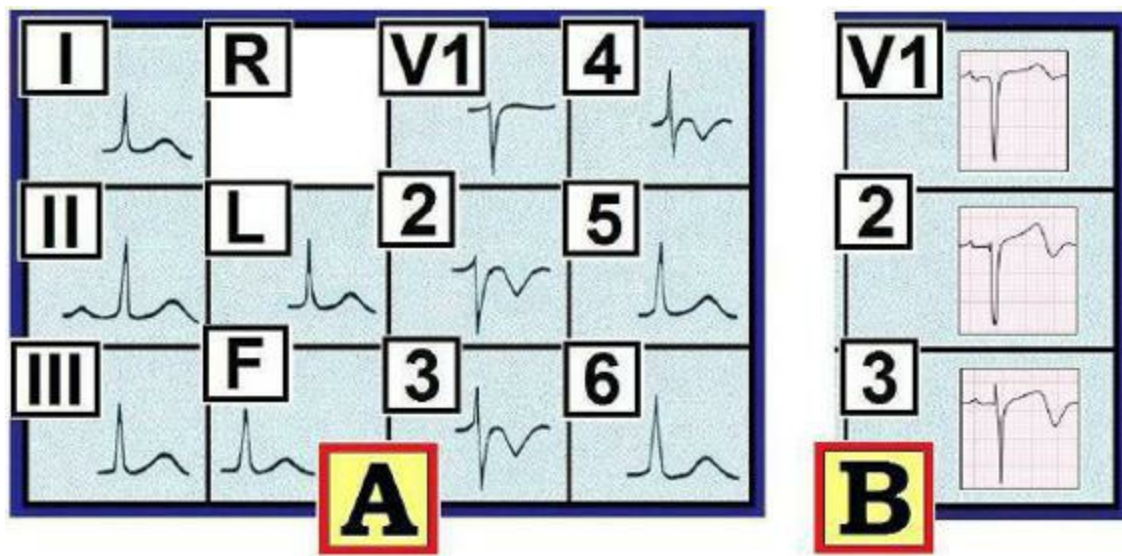


Figure 10.56-1: The ECG finding of *symmetric* anterior T wave inversion (as schematically shown in **Panel A**) — is *not* specific for a *proximal* LAD lesion. In contrast — the pattern seen in **Panel B** which shows slight ST elevation *with* a **steep T wave downslope** is *highly* specific for a critical **proximal LAD lesion**. OUR Preference: While fully acknowledging that *some* patients who manifest the T inversion pattern of Panel A will have coronary disease — We favor *reserving* the term, **“Wellens’ Syndrome”** for the ECG picture in **Panel B**, which in a *symptomatic* patient is *much more* specific for a proximal *critical* LAD narrowing.

10.57 – DeWinter T Waves



In follow-up to Wellens' Syndrome (*Section 10.54*) — the 2nd ECG pattern we highlight as being *highly specific* for the presence of a **critical proximal LAD narrowing** — is the presence of DeWinter T waves.

- Recognition of **DeWinter T waves** — is mandate for *immediate* intervention (*cardiac catheterization/acute reperfusion*).

10.58 – ECG Recognition: What are DeWinter T Waves?

Awareness of the relatively uncommon but highly characteristic DeWinter T wave sign is essential for *not* overlooking the *approximate 2%* of **acute anterior MI** patients who present with this ECG manifestation (*DeWinter, Wellens, Wilde: NEJM 359: 2071, 2008*).

- Rather than frank ST elevation that usually accompanies *acute* LAD occlusion — there is instead the unique **DeWinter complex**, with *upsloping J-point ST depression* *blending* into *very tall* upright **hyperacute T waves** in usually *several* of the precordial leads (*red arrows in leads V2, V3 of Figure 10-58.1*).

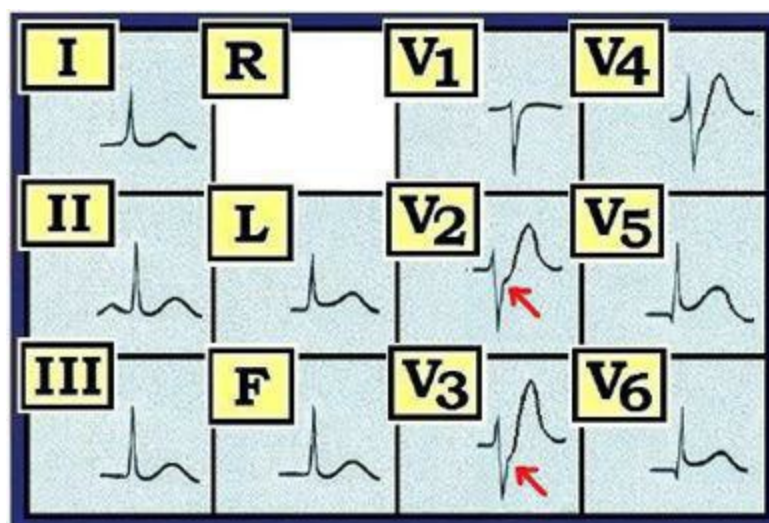


Figure 10.58-1: Schematic ECG from a hypothetical patient with *new-onset* chest pain. **DeWinter T waves** are seen in several of the precordial leads (*especially V2, V3 — but also lead V4*). Note disproportionately **tall upright (hyperacute) T waves** that arise from the characteristic **J-point ST depression** (*red arrows in V2, V3*). An encouraging sign that infarction has *not* yet taken place — is the presence of r waves with *appropriate* r wave progression in the anterior leads (*transition is slightly delayed — but the r wave does get progressively taller as one moves from lead V1-to-V4*). Prompt catheterization and reperfusion is essential!

10.59 – DeWinter T Waves: Clinical Characteristics

We emphasize the following key points about patients who present with chest pain and DeWinter T waves on ECG:

- Recognition of **DeWinter T waves** in patients with **new-onset chest pain** — is *highly* correlated with the finding of acute **proximal LAD critical** stenosis on cardiac catheterization.
- Rather than evolution of tall, peaked (*hyperacute*) T waves into frank ST segment elevation — the *DeWinter* T wave pattern is often surprisingly *static* over the ensuing few hours.
- *None* of the patients in the DeWinter series who manifested this ST-T wave pattern had acute left main occlusion on catheterization (*NEJM* 359: 2071, 2008). Thus, the DeWinter T wave complex appears **highly specific** for **proximal LAD stenosis** (*and not for a left main lesion*).
- *Despite* prompt recognition and intervention — a significant percentage of patients developed *positive* cardiac markers for acute infarction. One ECG sign that infarction may have *already* occurred — is development of an **anterior QS wave** or **loss of r wave amplitude** (See Figure 10.60-1 below).

10.60 – FIGURE 10.60-1: What is the “Culprit” Artery?

Consider the ECG shown in Figure 10.60-1 — obtained from a patient who presented to the ED with *new-onset* chest pain.

- Should the cath lab be activated for *acute* STEMI?
- IF so — What do you suspect the “**culprit**” artery is likely to be?
- How *many* ECG signs are present in Figure 10.60-1 to support your clinical impression?

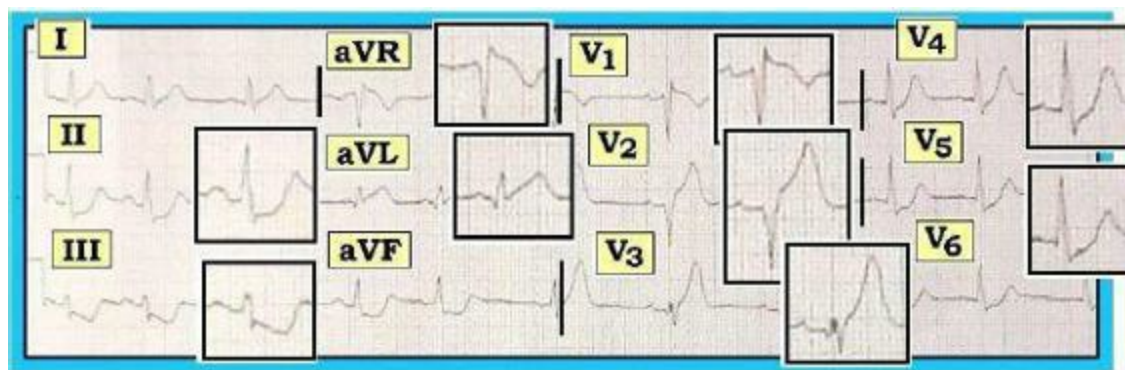


Figure 10.60-1: ECG obtained from a patient with *new-onset* chest pain. Should the cath lab be activated for **acute** STEMI? IF so — What is the “**culprit**” artery likely to be? (See text).

Answer to Figure 10.60-1: The rhythm is sinus. All intervals and the axis are normal. There is no chamber enlargement. There are however, a series of *alarming* findings that were recognized by the emergency team. Cardiac catheterization was performed *within* 15 minutes after this patient walked into the ED — with successful reperfusion of a **100% proximal LAD occlusion**. Clues to the need for *immediate* catheterization and clues indicating localization of the “**culprit**” artery to the *proximal* LAD including the following:

- **Hyperacute T waves** in *multiple* leads. These are best seen in leads aVL; and in V2,V3,V4.
- **DeWinter T Waves** — Rather than frank ST elevation (*as is usually seen with acute anterior infarction*) — there is 1-to-3 mm of **upsloping J-point ST depression** in one or more *precordial* leads that continue into **tall, positive symmetric T waves** (*seen best in leads V3,V4 of Figure 10.60-1*).
- Loss of anterior R waves (*with reduction in R wave amplitude between leads V1-to-V2*).
- Marked inferior **reciprocal ST depression**.
- **ST elevation** in leads aVR and V1.
- Incomplete RBBB (*rSr' in lead V1; narrow terminal S waves in leads I,V6*).

ECG PEARLS: In addition to precordial *DeWinter* T waves and loss of r wave between V1-to-V2 — there are a few **more signs** suggestive/consistent with an **acute proximal LAD lesion**. These include: **i)** Significant ST elevation in leads aVR and V1 (*with the amount of ST elevation in aVR not more than the amount in lead V1*); **ii)** Incomplete RBBB (*that is presumably new*); **iii)** Marked inferior *reciprocal* ST depression; and **iv)** The presence of a *hyperacute* T wave with ST elevation in lead aVL.

ACKNOWLEDGMENT: My appreciation to Andrew Bowman for allowing me to use the case and ECG from the patient whose tracing is shown in Figure 10.60-1.

10.61 – Takotsubo Cardiomyopathy



One of the most interesting new entities in clinical cardiology — is **Takotsubo Cardiomyopathy**. First described by Sato et al in 1990 as a reversible form of cardiac dysfunction — the precise mechanism of this intriguing clinical entity remains elusive. It is still *all-too-commonly* overlooked as a potential etiology for what otherwise might constitute *difficult-to-explain* ECG and clinical findings.

10.62 – FIGURE 10.62-1: *Acute STEMI — or Something Else?*

Consider the ECG shown in [Figure 10.62-1](#) — which was obtained from an older woman with severe *abdominal* pain. *No chest pain*. Serial troponins were no more than *minimally* elevated. She presented with heart failure.

- How might you explain this clinical picture?
- How can *definitive* diagnosis be made?

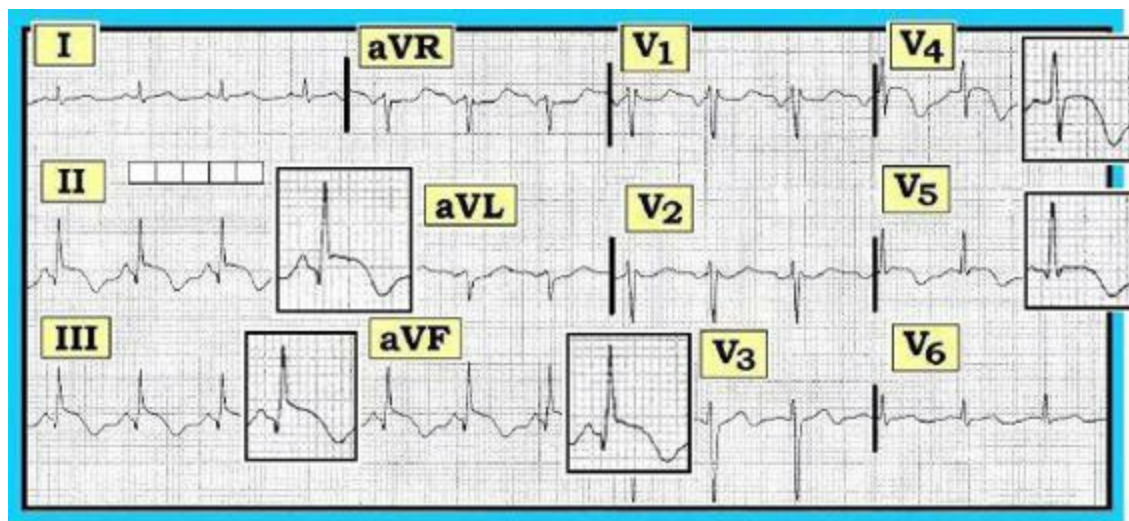


Figure 10.62-1: ECG obtained from an older woman with abdominal pain and evidence of heart failure. *No chest pain*. How might you explain this clinical picture? (See text).

Answer to Figure 10.62-1: The rhythm is sinus at ~100/minute. The PR and QRS intervals appear to be normal — but the QT is prolonged (*clearly more than half the R-R interval*). The axis is normal (+70 degrees). No chamber enlargement. An rSr' complex is noted in lead V1.

- Assessment of **Q-R-S-T Changes** — is remarkable for the presence of **inferior Q waves** — normal transition (*R wave becoming taller than the S wave between leads V3-to-V4*) — and **ST segment coving** with **marked ST elevation** in the **inferior leads**. This is accompanied by **deep T wave inversion**. Similar abnormal ST segment coving and elevation (*albeit not as marked*) is *also* present in **leads V4,V5**. Deep, *symmetric* T wave inversion that begins in lead V3 is seen

in V4,V5. *Reciprocal* ST depression is seen in lead aVL, and to a lesser extent in V1,V2.

Impression: Despite the absence of chest pain — the initial interpretation of this ECG was “probable acute STEMI (*ST-Elevation Myocardial Infarction*) in need of immediate cardiac catheterization for possible reperfusion”. After all — there are Q waves, ST segment coving and elevation, deep T wave inversion — and *reciprocal* ST depression. Acute RCA occlusion vs an LAD “*wrap-around*” lesion was suspected. However, cardiac catheterization revealed ***normal coronary arteries!***

- Instead of acute infarction — the ***ventriculogram*** revealed the diagnostic picture of apical ballooning with hypercontractility of the cardiac base that is characteristic of ***Takotsubo Cardiomyopathy***. The patient was treated supportively with recovery of left ventricular function over the next few weeks.

10.63 – *Takotsubo* CMP: *Clinical Features*

Takotsubo CMP (*CardioMyoPathy*) is an *underappreciated* cause of acute ECG abnormalities that are often accompanied by a degree of *new-onset* heart failure:

- First described in Japan in 1990 — the entity derives its name from a specially designed container used by Japanese fishermen to trap octopuses. The unusual *round* bottom and *narrow* neck design of ***takotsubo*** resembles the diagnostic picture on cardiac catheterization obtained as a result of *ballooning* of the cardiac apex with *hypercontraction* of the base (**Figure 10.63-1**).



Figure 10.63-1: Collection of actual takotsubo showing the round bottom and narrow neck — that resembles the *diagnostic* picture seen on the cardiac catheterization ***ventriculogram*** (*shown here during end-systole*). Note characteristic “*ballooning*” of the apex and *hypercontractility* of the base during cardiac cath (*See text*).

The precise **mechanism** for Takotsubo CMP remains elusive. While factors such as coronary spasm; disturbance of the microcirculation; coronary artery anatomic variation; and neurogenic myocardial stunning have all been implicated — their role is uncertain. What has been shown — is that **abnormal response** to an **increase** in **endogenous catecholamines** is almost always involved. Blood epinephrine and norepinephrine levels are *unmistakably* elevated (*sometimes dramatically*) in patients with Takotsubo CMP.

- In addition — there is *more-than-coincidental preceding occurrence* of **intense situational or psychological stress** in many patients. This may be in the form of severe **emotional** stress (*death of a loved one; break-up from significant other; overwhelming anxiety/depression*) — **pain** — and/or **fear** (*from earthquake or other catastrophic event*). It is easy to understand the rationale for *other* names that have been used for this syndrome (ie, **“Stress” Cardiomyopathy** or **“Broken-Heart” syndrome**).
- Although consensus is still lacking regarding specific criteria for defining Takotsubo CMP — there is general agreement that **cardiac cath** is **diagnostic** (ie, *apical ballooning but no “culprit” artery*).

SUMMARY of the Clinical Picture: Takotsubo CMP is most typically seen in an *older* patient (*most often in a post-menopausal woman*) — who presents with chest pain or *new* heart failure. Look for the following features:

- Likelihood of *preceding* severe *physical/emotional* stress.
- **Markedly abnormal initial ECG** — often with diffuse ST elevation in *inferior and antero-lateral* leads (*apical or LAD ‘wraparound’ distribution*). Associated T wave inversion *and/or* Q waves (*sometimes deep*) may be seen. Acute extensive *STEMI-in-evolution* is frequently the *initial* diagnosis (*as was the case in Figure 10.62-1*).
- Usually *no more* than **modest troponin elevation** (*troponins are often elevated — but generally not nearly as high as expected given how marked ECG changes are*).
- **Transient LV dysfunction** — which may be *severe* initially (*some patients present in pulmonary edema/cardiogenic shock requiring intra-aortic balloon pump support*). LV function *usually* resolves *within a few weeks*.
- Possibility of potentially *life-threatening* arrhythmias during the acute phase (*including VT/Torsades de Pointes*).
- *Absence* of pheochromocytoma, myocarditis or *other* underlying cardiac pathology to explain findings.
- **Generally favorable prognosis** (*often with full recovery within 1 month*) — but fatalities have been reported (*from LV free wall rupture; intractable pulmonary edema*).

NOTE: Variations on the above theme do exist. The syndrome of Takotsubo CMP is not limited to post-menopausal women — the ECG does not always show marked abnormality — and severe stress does not uniformly precede presentation.

- Anatomic areas *other than* the apex may also be affected. For example — there may be an

“*inverted takotsubo*” form, in which the apex is spared but the *base* of the heart is hypokinetic. The existence of such Takotsubo variants helps to explain the likely multifactorial etiology to this interesting syndrome.

Final Clinical PEARL: Think of the possibility of Takotsubo CMP when confronted with a patient who presents with a **markedly *abnormal* ECG** that ***doesn’t quite “fit”*** the clinical picture.

- ECG findings may be *out of proportion* to clinical findings. ECG changes may involve several lead areas (*especially inferior and anterior precordial leads* — *which typically assess the cardiac apex*).
- There may be an element of heart failure. Serum troponins may be positive.
- The patient is usually an older adult (*especially a post-menopausal woman*).
- There has usually been some form of severe *preceding* “stress”.

Duchenne Becker ➞ **Muscular Dystrophy**

Among *non-ischemic* etiologies for markedly **abnormal** ECGs in young individuals — is the series of **muscular dystrophies**. The best known and most common of these is **Duchenne Muscular Dystrophy (DMD)** — but there are more than 20 different genetic forms of these rare muscular dystrophy disorders. DMD occurs in ~1/3,000 boys (*it is an X-linked disorder*) — with unfortunate outcome of progressive muscle deterioration leading to death at an early age.

- Among the many other forms of muscular dystrophy is **Becker Muscular Dystrophy (BMD)** — which is felt to be a *less* severe form than DMD. There are similarities among the different forms of muscular dystrophy (*re being genetic defects leading to progressive muscle weakness/deterioration*) — though variations exist depending on muscle groups affected and prognosis.
- The defect in DMD is the absence of a critical muscle protein (*ergo rather than “dystrophy” — it is really a myopathy*).
- Symptoms of DMD typically begin before age 5 (*sometimes in infancy*). The boy with DMD will lag in development. He may be clumsy, unbalanced, weaker and easily prone to fatigue.
- “Pseudohypertrophy” (*enlargement of calf and deltoid muscles*) occurs — and may be an early sign (*due to replacement of injured muscle cells by fibrous or scar tissue*).
- Progressive muscle weakness develops — beginning proximally (*legs, pelvis*) before spreading to other areas.
- Diagnosis of DMD (*and other muscular dystrophies*) is suspected by increased CK levels on lab testing — and confirmed by muscle biopsy and genetic testing.
- **DMD is uniformly fatal.** The disorder is progressive and ultimately affects all voluntary muscles. The heart and respiratory muscle groups are typically affected by the teens. Myocardial deterioration (*cardiomyopathy*) and respiratory failure represent the major threat to life. There is no cure (*most boys die before age 25*).
- Prognosis in *other* forms of muscular dystrophy is not necessarily uniformly fatal as it is in DMD. Cardiac involvement (*and ECG changes*) are common in many (*but not all*) of these other forms. Females may be affected in some of the other forms.
- **BOTTOM Line:** It is well to be aware of the group of muscular dystrophies as one of the causes of a **markedly abnormal ECG** in a **young adult** (*especially in males*). Given the variation in severity and presentation — the diagnosis of a form of muscular dystrophy might *not* always be known at the time an ECG is done.

10.65 – Muscular Dystrophy: Common ECG Abnormalities

By young adulthood — the ECG with DMD (*and with many other forms of muscular dystrophy*) will usually be abnormal. Among the **ECG abnormalities** that are commonly seen include the following:

- **Abnormal Q waves** not due to infarction. These are most often seen in *lateral* leads.

- Sinus tachycardia (*due to impaired cardiac function*).
- **RBBB** — that often manifests a peculiar **polyphasic rsr'** or rsr's' in lead V1.
- **Tall anterior R waves** — that may manifest surprisingly high amplitude. This may result in *early transition* or even as a *Tall R* in V1 (*Section 10.47*).
- *Other* conduction defects may be seen — including **nonspecific IVCD** with *unusual* QRS morphology.

BOTTOM Line: The ECG of a patient with muscular dystrophy may be markedly abnormal. This should be easy to understand given the genetic fault in muscle protein (*with replacement of myocardial tissue by fibrous and scar tissue*). This also explains the unusual array of ECG abnormalities and conduction defects that may be seen.

10.66 – FIGURE 10.66-1: *Abnormal ECG in a Young Subject*

Consider the ECG shown below in Figure 10.66-1 — obtained from a 22-year old man in a wheelchair because of longterm disability and weakness. *No chest pain*.

- How would you interpret this ECG?
- Can you think of a clinical entity that might account for this clinical scenario?

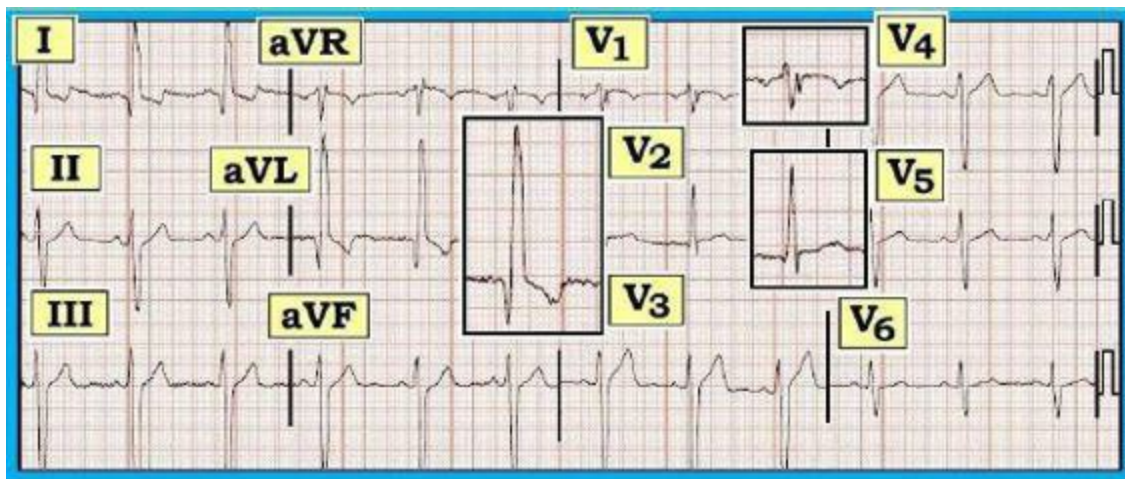


Figure 10.66-1: 12-lead ECG obtained from a 22-year old man in a wheelchair with longterm disability. What medical diagnosis might explain this clinical scenario in association with this ECG?

Answer to Figure 10.66-1: The ECG shows sinus rhythm at ~70/minute. The PR interval is normal — but the QRS is prolonged to *at least* 0.12 second. QRS morphology in the 3 key leads (*I, V1, V6*) is *not* consistent with either left or right bundle branch block. Therefore — We would classify the conduction defect as **nonspecific IVCD** (*IntraVentricular Conduction Delay*).

- Continuing with the interpretation — We note **marked LAD** (*Left Axis Deviation*) that is consistent with **LAHB** (*Left Anterior HemiBlock*) in view of the predominantly *negative* QRS complex in lead II.
- QRS amplitude is *markedly* increased in lead aVL — although reliability of the ECG diagnosis of **LVH** is reduced in the presence of conduction defects.

- Regarding **Q-R-S-T Changes** — there are deep and wide **Q waves** in the **high lateral leads** (*I, aVL*). In addition — there is a small q wave in lead V2.
- QRS morphology in **lead V1** is peculiar — as there is an **rSr's' complex**. This is followed by abrupt **early transition** that takes place between lead V1-to-V2. A *disproportionately tall R wave* is noted in **lead V2**.
- R wave amplitude drops off by lead V3 — with persistent S waves seen throughout the remaining precordial leads.
- There is **ST depression** in *lateral* leads I and aVL, which may reflect **LV “strain”** from suspected LVH. That said — ST-T wave changes do *not* appear to be acute.

IMPRESSION: This ECG is clearly abnormal and highly *unusual* for a young adult. The diagnosis of a form of muscular dystrophy would explain *all* of the abnormalities seen (*Section 10.65*).

ACKNOWLEDGMENT: My appreciation to Dr. Harsha Nagarajarao for allowing me to use the case and ECG from the patient whose tracing is shown in Figure 10.66-1.

10.67 – Hypothermia (*Osborn Wave*)



The **Osborn Wave** — was first described in 1953 by JJ Osborn. The wave is commonly linked to **hypothermia** — but *other* entities (*including CNS injury and ventricular fibrillation*) may also be associated with it. A number of *other* names have been attributed to this ECG finding (*“camel-hump” sign; hypothermic wave; prominent J wave*).

- The **Osborn Wave** — is a *positive* deflection that occurs *just after* the QRS and at the *beginning* of the ST segment (**Figure 10.67-1**).
- Osborn waves are often *not* seen until the temperature drops *below* -32 degrees Centigrade (=89.6 degrees Fahrenheit).
- Other commonly **associated** ECG features with **Hypothermia** include: **i)** Bradycardia (*which may be marked*); **ii)** Atrial fibrillation; and **iii)** Artifact (*from baseline undulations resulting from associated shivering*).

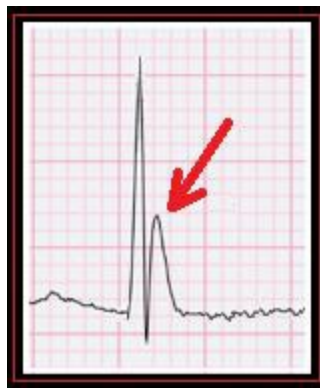


Figure 10.67-1: Osborn wave — highlighted by the *red* arrow. The ST segment is flat. Note fine undulations in the baseline that are commonly seen with hypothermia from associated shivering.

10.68 – FIGURE 10.68-1: *ECG Features of Hypothermia*

Consider the ECG shown below in **Figure 10.68-1** — obtained from a homeless man who was stuporous and intoxicated when found in an open wet field.

- What would you guess this patient’s core temperature to be?

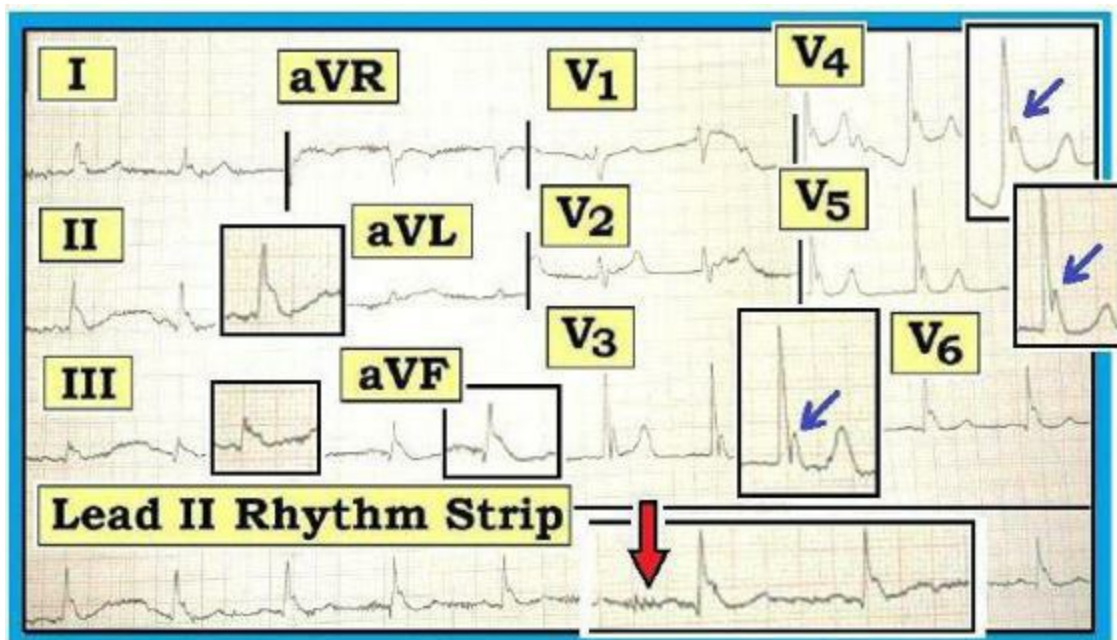


Figure 10.68-1: ECG obtained from a homeless man who was stuporous and intoxicated when found in an open wet field. Prominent **Osborn Waves** are noted in *multiple* leads (*blue arrows*). Note fine undulations in the baseline from *associated shivering* (*large red arrow in lead II rhythm strip*).

Answer to Figure 10.68-1: There is **baseline artifact**. We suspect the rhythm is sinus based on what appear to be low amplitude P waves with a fixed PR interval in lead II (*and overall regularity of the rhythm*) — but it is difficult to be sure of atrial activity given the artifact. Frequent occurrence of atrial fibrillation in hypothermic patients is another potential reason for baseline undulations (*and another reason rhythm determination may be challenging*).

- **Osborn Waves** are present. These are *especially* marked in leads V3,V4,V5 (*blue arrows*) — in which they attain *at least* 5 mm in amplitude! Osborn waves are also seen to a lesser degree in leads V2 and V6. They may also be present (*hiding in the terminal portion of the QRS complex*) in the inferior leads — but the distinct notching of an Osborn wave is absent in these leads.
- Small inferior q waves are seen — but ST-T wave changes do *not* appear to be acute.
- T waves appear prominent and somewhat peaked in V3,V4,V5.

Clinical Correlation: The patient's core temperature on arrival was 30 degrees Centigrade (86 degrees Fahrenheit). Serum K+=7.3 mEq/L; arterial pH=6.89. Tremor (*producing baseline artifact as seen here*) is common in hypothermic patients. Of interest — Osborn waves resolved as the patient was warmed. Despite tropical location (*Costa Rica*) — this case illustrates that hypothermia may occur in warm weather places if predisposing circumstances are present (*in this case alcohol intoxication and poor choice of shelter*).

ACKNOWLEDGMENT: My appreciation to Leonardo Chacon for allowing me to use the case and ECG from the patient whose tracing is shown in Figure 10.68-1.

11.0 – Electrolyte Disorders



Electrolyte Disorders

With possible exception of hyperkalemia — an “*electrolyte disorder*” will rarely be the *primary* indication for obtaining an ECG. That said — the ECG may be of *definite* assistance in assessing *some* patients with *certain* types of electrolyte disorders, especially for explaining some of the ST-T wave abnormalities that may be seen. Consider the following:

- **Potassium** — There is a surprisingly **good correlation** between serum K⁺ (*potassium*) levels and the ECG when serum K⁺ is **elevated**. There are times when the ECG will be completed *before* blood results are back — strongly suggesting *not only* the diagnosis of hyperkalemia, but its *likely* severity (and the need for immediate treatment even before serum value confirmation). Unfortunately — correlation between serum levels and ECG changes is *poor* when serum K⁺ is normal or low (*Section 11.7*).
- **Magnesium** — is still *all-too-often* the “forgotten” cation. Hypomagnesemia is common when serum K⁺ is low. **Low serum Mg⁺⁺ (magnesium) levels** produce virtually **identical ECG changes** as **hypokalemia** (*Section 11.8*). On the other hand — **Hypermagnesemia** usually does *not* produce ECG changes until serum Mg⁺⁺ levels are *markedly* increased (*usually* >8-10 mEq/L) — at which time there may be bradycardia, *prolongation* of PR/QRS/ QT intervals and/or AV block. *Clinical* hypermagnesemia to this degree is distinctly uncommon in clinical practice unless there is renal failure with Mg⁺⁺ supplementation.
- **Calcium** — either in excess or deficiency, will *not* often produce *recognizable* effects on the ECG. That said — there are changes to look for: **i) Hypocalcemia** — typically *prolongs* the QT interval *without* affecting the subsequent ST segment; and **ii) Hypercalcemia** — may *shorten* the QT (*Section 11.1*).
- **Other Electrolyte Disorders** (*including abnormalities in serum Sodium and Phosphorus*) — in our experience do *not* produce any specific (*recognizable*) ECG picture. That said, there will often be *more* than a single electrolyte abnormality at a time — so *additive* effects are possible.

In this segment — We *limit* discussion and illustration of ***electrolyte-induced* ECG Changes** to the following:

- Calcium disorders — ***Section 11.1***.
- Hyperkalemia — ***Section 11.3***.
- Hypokalemia — ***Section 11.7***.
- Hypomagnesemia — ***Section 11.8***.
- Discussion of **U waves** — ***Section 11.9***.

11.1 – CALCIUM: ECG Changes of Hyper- & HypoCalcemia

As noted above — it will *not* be often that you will be able to look at an ECG and say, “*These ECG*

changes are due to an excess or deficiency in serum Calcium”. There are **several** reasons for this: i) ECG changes of hypo- and hypercalcemia are both *subtle* and often do *not* occur until excess or deficiency of this cation is marked; and ii) Calcium disorders are commonly associated with *other* metabolic and/or clinical problems. That said — it is still useful to be aware of the effect that Calcium disorders may have on the ECG. Consider the following:

- **HypoCalcemia** — typically *prolongs* the **QT interval**. As a result — a *low* serum Calcium level is one of the causes in our **LIST #3** for a **long QT** (*Section 06.2*). In theory — the ST segment is *not* affected by hypocalcemia, such that the T wave at the end of the *long* QT looks normal. This effect is *schematically* illustrated in **Figure 11.1-1** — in which compared to the situation when serum Calcium and the QT interval are *both* normal (**Panel A**) — a seemingly *unaltered* T wave is delayed in appearance by the *long* QT of hypocalcemia (**Panel B**). Clinically — the ECG picture seen in **Panel B** is typically *not* seen until serum calcium is *markedly* decreased. Even then, it may be difficult to distinguish the ECG effect of low serum calcium from that of other associated electrolyte disorders. **Bottom Line:** *Don't expect* to recognize hypocalcemia on ECG.
- **Panel C** — Be aware that **Hypocalcemia and Hyperkalemia** may occur *together* in patients with renal failure. Clinically — this *combined* electrolyte disorder may occasionally be suspected by the ECG finding of peaked (*pointed*) T waves with *narrow* base that occur at the end of a *long* QT (**Panel C** in **Figure 11.1-1**).
- **HyperCalcemia** — may *shorten* the **QT interval**. That said — *Don't expect* to see QT shortening *until* serum Ca^{++} levels are *markedly* increased (*usually to more than 12mg/dL*). Even then — it will often be *exceedingly* difficult to distinguish between a QT interval that is normal (**Panel A** in **Figure 11.1-1**) — *vs* a QT that is “short” *with* *early peaking* of the T wave (*as is theoretically seen with hypercalcemia* — **Panel D**).

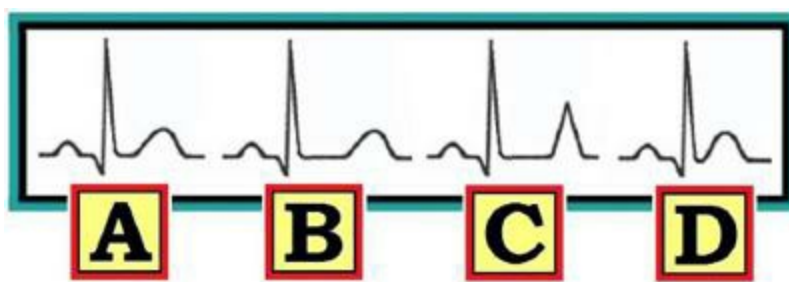


Figure 11.1-1: *Schematic illustration of ECG changes with Calcium disorders. **Panel A** — the ST-T wave and the QT interval are both normal. **Panel B** — Hypocalcemia, which may lengthen the QT interval. The ST-T wave is otherwise unaffected. **Panel C** — Hypocalcemia *with* Hyperkalemia, as may occur with renal failure. The tall, peaked (*pointed*) T wave with *narrow* base of hyperkalemia — is *delayed* by the *long* QT of hypocalcemia. The preceding long ST segment is flat. **Panel D** — Hypercalcemia, which may *shorten* the QT (*with reduced time until T wave peaking*). Clinically — it is usually difficult to distinguish between a *normal vs short* QT interval (See text).*

11.2 – **Figure 11.2-1: Acute STEMI or HyperCalcemia?**

Consider the ECG shown below in **Figure 11.2-1** — obtained from a 60-year old man being treated for *advanced* lung cancer. He presented to the ED (*Emergency Department*) with weakness and

palpitations, but *no* chest pain.

- Should the cath lab be activated for *acute* STEMI?
- If so — what do you suspect the “culprit” artery is likely to be?
- Is *anything else* likely to be going on in **Figure 11.2-1** — in view of this clinical history?
- **HINT #1:** Be *systematic* in your interpretation (*including assessment of all intervals*).
- **HINT #2:** Feel free to review Section 11.1 that was just covered *before* formulating your answer.

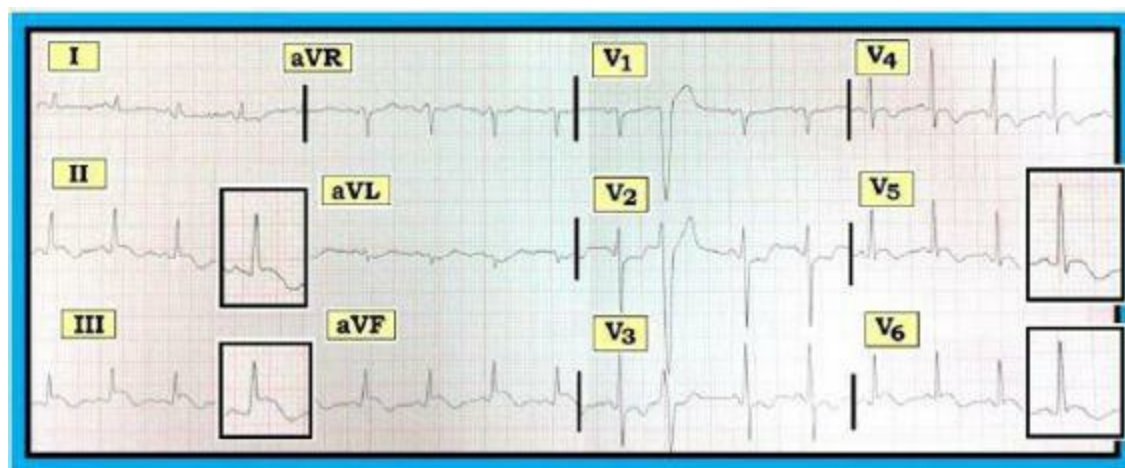


Figure 11.2-1: ECG obtained from a 60-year-old man with lung cancer, weakness and palpitations — but *no* chest pain. Is this ECG diagnostic of an *acute* STEMI? Is *anything else* likely to be going on? (**HINT:** Feel free to review Section 11.1 *before* formulating your answer).

Answer to Figure 11.2-1: The rhythm is sinus tachycardia. One PVC (*Premature Ventricular Contraction*) is seen (*the early wide-complex beat in simultaneously recorded leads V1,V2,V3*). The PR and QRS intervals are normal. The **QT interval** is *difficult to assess* given the tachycardia and lack of distinct end point of T wave inversion. The axis is normal at +70 degrees. No chamber enlargement.

- Regarding **Q-R-S-T Changes** — there are no definite q waves; transition is normal (*occurring between leads V2-to-V3*). The most remarkable finding relates to ST-T wave changes.
- There is **coved ST elevation** in *each* of the *inferior* leads (II,III,aVF) — and in *lateral* precordial leads (V4,V5,V6). In *each* of these leads, there appears to be **T wave inversion** following descent of the ST segment.
- There is **reciprocal ST depression** in leads aVL, V2, and to a lesser extent in lead V3. Lead V3 shows transition between the *flat* ST depression in lead V2 — and the ST coving and T wave inversion that begins in lead V4.

Initial Impression: The ECG in Figure 11.2-1 suggests **acute STEMI** (*ST Elevation Myocardial Infarction*) — in that there is *coved* ST elevation with T wave inversion and *reciprocal* ST depression. That said — there are **3 elements** about the history given and the ECG in **Figure 11.2-1** that should be cause for contemplation: **i)** There is no history of chest pain; **ii)** The patient has

advanced lung cancer; and **iii**) The *shape* of the ST segment elevation is a bit “off” for acute STEMI. **KEY Clinical Point:** Despite *valid* concern about possible acute *infero-postero-lateral* STEMI (*either from a proximal right coronary vs dominant left circumflex occlusion*) — the *lack* of chest pain and *lack* of any defined “onset” of symptoms in the context of a patient with *advanced* cancer should prompt **additional data gathering** *prior* to cath lab mobilization.

- Initial serum troponins were negative.
- The patient was taken to the cath lab. *No* acute lesion and *no* significant coronary disease was found.
- While in the cath lab — *additional* lab values returned showing a *markedly* elevated **serum Calcium** value = **17 mg/dL**.
- *Comparison* ECGs were ultimately found. These were clearly abnormal. While a similar degree of ST elevation was *not* seen on prior tracings — there was definite ST segment coving with T wave inversion present on earlier tracings.

Clinical SYNTHESIS: This case is admittedly subtle and complex. It is clearly *beyond-the-core* for the beginning interpreter. Nevertheless — We feel it is an excellent teaching example for emphasizing a number of clinical points that are of interest for providers of any level. These include the following principles:

- **All ST segment elevation** is not necessarily the result of **acute coronary occlusion!** In addition to common “*other causes*” of ST elevation (ie, *early repolarization variants*; *LVH*; *acute pericarditis*) — chronic ST elevation may result from either ventricular aneurysm or cardiomyopathy. This case serves to remind that **Hypercalcemia** is yet another **potential STEMI-mimic**.
- Textbooks describe **QT interval shortening** as the principal ECG manifestation of **hypercalcemia**. That said — recognition that the QT interval is “short” is not easy to detect because: **i**) Usually *marked* hypercalcemia (*levels >12.0 mg/dL*) are needed before the QT noticeably shortens; **ii**) it will often be difficult to distinguish clinically between a QT interval that is within the “normal” range vs one that is “too short”; and **iii**) ECG manifestations of hypercalcemia are *superimposed* on any baseline abnormalities that may be present.
- We mentioned that the **shape** of the **ST elevation** in this case was a bit “off” for *acute* STEMI. By this — we mean that the *coved* ST segments seemingly peak a tad *earlier* than is usually seen. That said — the *overall* QT interval in this case is *not* shortened. If anything — the overall QT interval is *lengthened* by *preexisting* T wave inversion that was seen to be present on *prior* comparison tracings.

BOTTOM Line It is virtually **impossible** to **attain 100% accuracy** in knowing when to **activate** the **cath lab** based on assessment of a *single* initial ECG. Centers that boast an accuracy rate of 100% are clearly *missing* a certain percentage of *acute* MI patients who would probably benefit from early reperfusion.

- Occasional *false* activation of the cath lab is inevitable — and clearly *understandable* in this

case given the ECG picture of ST elevation with T wave inversion and *reciprocal* ST depression. “**Hindsight is 100% in the retrospectoscope**” (Grauer — *circa 1980*). That said — the lesson to be learned is that the ECG is not a perfect tool — and the patient in this case gave *no* history of chest pain, and *no* defined onset of symptoms. There are times when *slight* delay before activating the cath lab is appropriate so that some *additional* evaluation can be performed.

ACKNOWLEDGMENT: My appreciation to Jiann Ruey Ong for allowing me to use the case and ECG from the patient whose tracing is shown in Figure 11.2-1.

11.3 – HYPERKALEMIA: ECG Manifestations/Clinical Features

The ECG may prove invaluable in *expediting* recognition of clinically *important* hyperkalemia. This is because with Hyperkalemia — there is a surprisingly **close correlation** between **serum K⁺ levels** and ECG findings. We highlight this relationship in *schematic* Figure 11.3-1:

- **Panel A** — the serum K⁺ level is normal (*between* ~3.5-5.0 mEq/L). The QRS is of normal duration. The T wave is smooth.
- **Panel B** — shows *peaking* of the T wave, which is generally the *earliest* change of hyperkalemia (K⁺ ~5.5-6.5 mEq/L). This may initially be subtle.
- **Panel C** — The **T wave** becomes **taller** and **more peaked** (K⁺ ~6.5-7.5 mEq/L). With progressive hyperkalemia — the T wave takes on an “**Eiffel Tower**” appearance. Not only is the T wave tall and peaked — but it manifests a **narrow base** (*like the Eiffel Tower*). This is in contrast to the T wave that is sometimes seen in *healthy* individuals as a **Normal Variant** (*lower right insert within **Figure 11.3-1***) — in which the T wave is rounded, its sides are not symmetric (*slower ascent than descent*) — and the T wave has a *broader* base (*Discussed momentarily in Section 11.6*).
- **Panel D** — P wave amplitude decreases; the PR interval lengthens — and the QRS widens (K⁺ >8 mEq/L). Recognition of these ECG features signals potentially *life-threatening* hyperkalemia.
- **Panel E** — P waves often *disappear* at serum K⁺ levels >8-9 mEq/L. Yet *despite no longer* seeing P waves on the surface ECG — the rhythm is *still* initiated by SA nodal impulses (**sinoventricular rhythm**). The result may be a *wide-QRS* rhythm without P waves that is *still* supraventricular (*due to associated hyperkalemia* — See Section 11.4). Eventually (*at serum K⁺ levels >9-10 mEq/L*) — the QRS complex becomes *sinusoid*. Ventricular fibrillation *and/or* asystole usually follow.

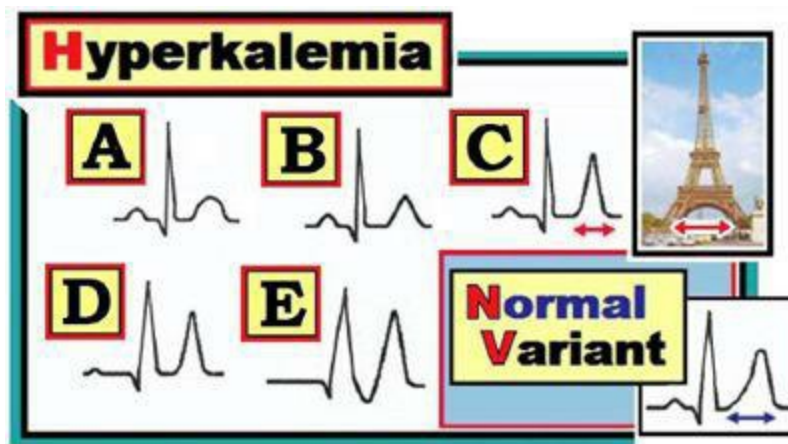


Figure 11.3-1: ECG Manifestations of *Hyperkalemia*. Starting from a normal T wave (**Panel A**) — the T wave becomes progressively *taller* and *more peaked* from **Panel B** ($K^+ \sim 5.5-6.5 \text{ mEq/L}$) to **Panel C** ($K^+ \sim 6.5-7.5 \text{ mEq/L}$) as it takes on an “Eiffel Tower” appearance. **Panel D** — P wave amplitude decreases; the PR interval increases; the QRS widens ($K^+ > 8 \text{ mEq/L}$) — until the *pre-lethal* stage in **Panel E** ($K^+ > 8-9 \text{ mEq/L}$) with *sinoventricular* rhythm (*P wave no longer seen*); marked QRS widening; and ultimately a *sinusoidal* pattern prior to VFib or asystole. Note the difference in shape between the T wave in **Panel C** — *vs* the **Normal Variant** pattern (lower right insert — *asymmetric T wave; rounder; with a wider base*).

Clinical NOTE: At the bedside — our *first* inclination when told of an *elevated* K^+ level — is to *validate* the **reading** (and *rule out hemolysis*). We do this by *repeating* the **blood test**.

- At times — simply obtaining an **ECG** may be a *faster* way to determine IF there is cause for concern. The *absence* of ECG signs shown in **Figure 11.3-1** — suggests that serum K^+ is probably *not* significantly elevated.

Increasing Incidence of HyperKalemia: Clinically — the incidence of Hyperkalemia continues to increase. This is *at least in part* because of the *increasing* number of patients on longterm dialysis.

- More patients have diabetes than ever before (*obesity epidemic in the general population*). These patients are living longer — so *more* of them than ever before go on to eventually **require dialysis**. As a result — virtually all health care providers will encounter patients with hyperkalemia at some frequency.
- **KEY Clinical Point:** With rare exceptions (ie, *hyperkalemic periodic paralysis*) — Hyperkalemia does *not* usually develop unless there are one or more **predisposing factors**. These should be inquired about in the history. Potential *predisposing* factors may include: **i)** Use of K^+ -retaining medications (*ACE-inhibitors; angiotensin-receptor blockers; K^+ -sparing diuretics*) ; **ii)** Potassium supplementation; **iii)** Renal failure/dialysis; **iv)** Acidosis; **v)** Dehydration; and **vi)** *Severe trauma*.

11.4 – Figure 11.4-1: *Ventricular Rhythm vs Hyperkalemia?*

Consider the 2 rhythm strips shown below in **Figure 11.4-1**. Imagine each tracing was obtained from

a hemodynamically stable older adult.

- Is the rhythm in **Panel A** *ventricular* escape?
- Is the rhythm in **Panel B** a *slow* form of VT?
- How would you proceed *clinically* in *each* case?
- What clinical information would you want to know about each patient? (**HINT:** *How might these 2 rhythms relate to the content of Section 11.3?*).

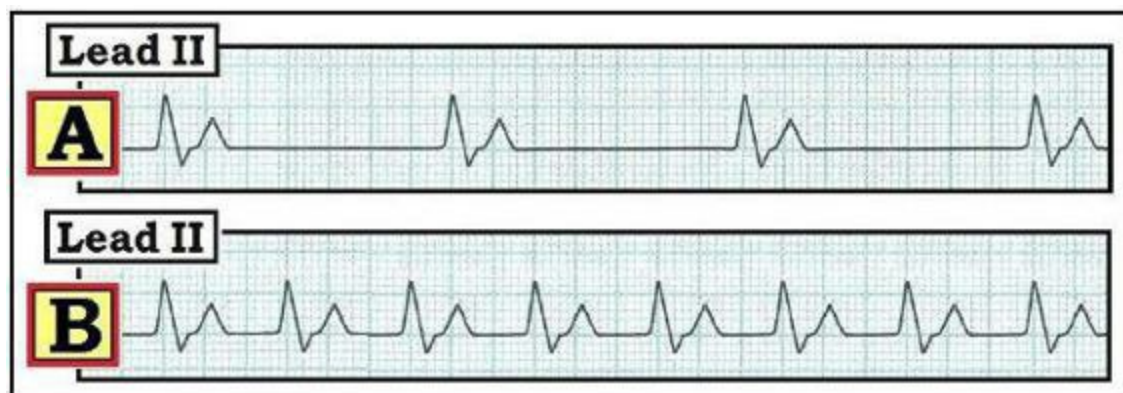


Figure 11.4-1: *Ventricular rhythms vs hyperkalemia? (See text).*

Answer to Figure 11.4-1: Although our *initial* impression for *both* arrhythmias in this Figure is that a **ventricular rhythm** is present in *each* case — this is *not* necessarily the correct answer. For example — IF told that each patient was hemodynamically stable and on *longterm* dialysis (or *had a history of chronic kidney disease*) — then a **stat serum K⁺** value becomes *essential* for accurate diagnosis and *optimal* management.

- **Rhythm A** (in Figure 11.4-1) — shows QRS *widening* — a *slow* ventricular rate (~45/minute) — *lack* of P waves — and suggestion of T wave peaking. In a patient on longterm dialysis — one has to consider **hyperkalemia**!
- **Rhythm B** — closely resembles AIVR (*Accelerated IdioVentricular Rhythm*) — as was discussed in Section 02.38. But IF this patient had renal failure — **Hyperkalemia** would *again* become a key consideration.

BOTTOM Line: IF it turned out that *marked* hyperkalemia was the reason for the *wide* rhythms in Panel A and Panel B — then *rather than* Pacing/Atropine — the treatment of choice *would be* Bicarb/Calcium.

- **History is *ever* important!** Sometimes a *stat* K⁺ level is needed for accurate diagnosis. Given the hemodynamically *stable* condition of each patient in Figure 11.4-1 — there *is* time for lab confirmation of the diagnosis.
- Knowing that a hemodynamically *stable* patient with a *wide-QRS* rhythm *without* sinus P waves has **renal failure** — goes a long way toward suggesting that what we are seeing in Figure 11.4-1 is a rhythm in a patient with **hyperkalemia** (*with changes in QRS morphology corresponding to that seen in Panel D or Panel E from Figure 11.3-1*). This is important since *optimal*

treatment of this patient depends on *accurate* diagnosis of the reason for QRS widening.

11.5 – Figure 11.5-1: *Ischemia vs Hyperkalemia?*

Consider the ECG shown in **Figure 11.5-1**. Clinically — What is *likely* to be going on? Is there ECG evidence of *ischemia*?

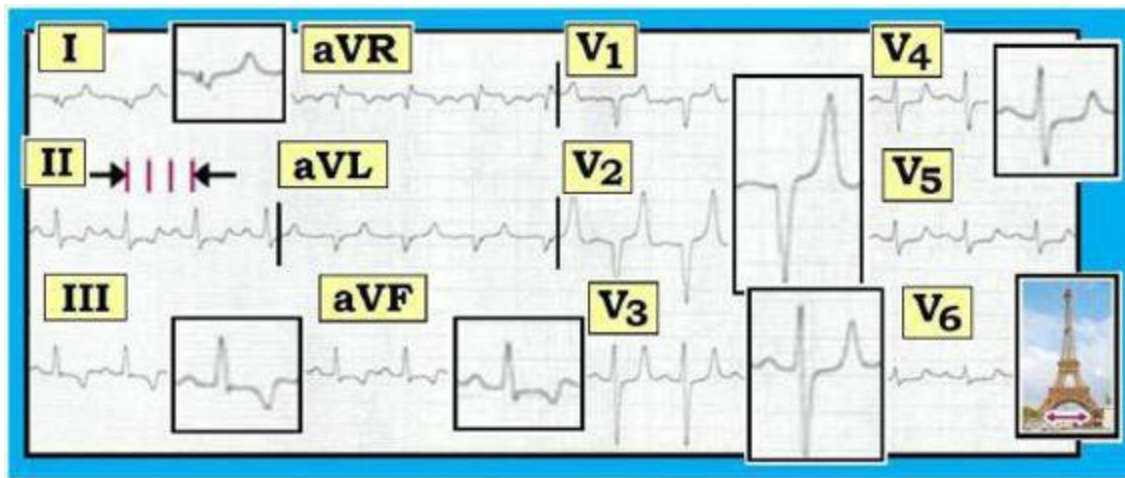


Figure 11.5-1: *Ischemia vs hyperkalemia?* (See text).

Answer to Figure 11.5-1: Sinus rhythm at ~100/minute. The PR interval is normal; the QRS and QT are *borderline* prolonged. *Rightward* axis. *No* chamber enlargement.

- **QRST Changes** — There is a Q wave in lead I and there are QS complexes in leads aVL, V1, V2. Transition is slightly delayed (*occurs between V4-to-V5*). The most remarkable finding is the very **tall, peaked T waves** with *narrow* base (“**Eiffel Tower**” effect) in at least *several* precordial leads. This strongly suggests **hyperkalemia** — perhaps with a **serum K⁺ ~6-7.5 mEq/L** (See Figure 11.3-1). In addition, T waves are *inverted* sharply in leads III and aVF and there is some ST *depression* in *infero-antero-lateral* leads.
- We would want to inquire from the history IF there were **predisposing factors** (*such as renal failure*) to the *severe* hyperkalemia that we suspect from this tracing.

Clinical NOTES: The ECG is the **net result** of the heart’s electrical activity. Therefore — ECG changes from *new* hyperkalemia will be *superimposed* on any *preexisting* ST-T wave abnormalities.

- We’ll have to *wait* until hyperkalemia is corrected *before* knowing IF *abnormal* findings (*right axis; QS complexes; T wave peaking; ST depression and T inversion*) are due to ischemia *as well as* hyperkalemia.
- In our experience — We have seen a great variety of *abnormal* ECG findings *disappear* once hyperkalemia is corrected. For unknown reasons — marked right axis (*as seen in Figure 11.5-1*) often resolves once serum K⁺ is again normal.
- Note in Figure 11.5-1 — that the *negative* peak of the *inverted* T waves in leads III, aVF is sharp (*pointed*). Although *positive* peaked and pointed T waves are much more commonly associated with hyperkalemia — T wave peaking may occasionally be *negative* with this electrolyte

disorder. We'll only know for sure after serum K⁺ is corrected and a *repeat* ECG is obtained.

- **BOTTOM Line:** Be sure to *repeat* the ECG after hyperkalemia is *corrected* and *correlate* tracings to the *clinical* situation (ie, *chest pain with possible ischemia* vs *simple hyperkalemia?*).

11.6 – Figure 11.6-1: Hyperkalemia vs Normal Variant?

Finally, consider the ECG in **Figure 11.6-1** — obtained from a *healthy* 30-year old man. How might your interpretation *change* — IF the patient was *older* on *longterm* dialysis?

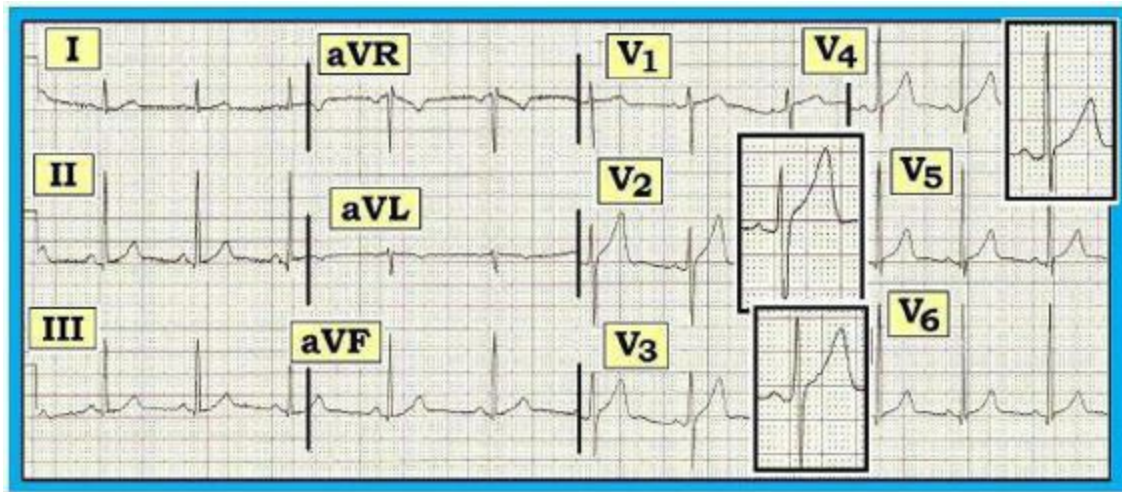


Figure 11.6-1: Hyperkalemia vs Normal Variant? (See text).

Answer to Figure 11.6-1: Sinus arrhythmia. Normal intervals and axis. No hypertrophy (*since the patient is only 30 years old*). T waves are peaked — and there is slight J-point ST elevation with an upward concavity (“smiley” configuration) in several precordial leads. **IMPRESSION:** Given that the patient is a young, otherwise healthy adult — this ECG almost certainly represents **ERP** (*Early Repolarization Pattern* — Section 09.02).

- But IF the patient was *older* with chronic kidney disease — then we would *definitely* **check** the *serum K⁺ value*. Even though T waves here do not manifest the typical “Eiffel Tower” effect in that they had a *broad* base with *asymmetric* ascent and descent of the T wave (**Figure 11.6-2**) — all bets would be off IF the patient had renal disease or other potential reason for hyperkalemia. **BOTTOM Line:** Clinical correlation is *key* to optimal interpretation.



Figure 11.6-2: Comparison of ECG characteristics for the T wave of hyperkalemia (**Panel C** —

reproduced from Figure 11.3-1) — vs what is commonly seen as a **normal variant** in otherwise healthy adults. Note that the T wave of **Hyperkalemia** (in Panel C) generally manifests a more peaked (*pointed*) T wave with *narrow* base and *symmetric* ascent/descent of T wave. In contrast — the T wave in a **normal repolarization variant** tends to be *rounder* with *wider* base and *asymmetric* ascent/descent of T wave. Exceptions exist — so *clinical* correlation is essential (*See text*).

11.7 – HYPOKALEMIA: ECG Manifestations/Clinical Features

In contrast to hyperkalemia — the ECG is not a reliable tool for assessing **hypokalemia**. While true that it is *unlikely* for the ECG to be “normal” IF clinically *significant* hypokalemia is present — **ECG changes** are often **nonspecific** — they may be *difficult-to-interpret* — and in our experience, ECG changes are *suboptimal* in ability to correlate with degree of severity. We highlight the following **Clinical Caveats** regarding **ECG assessment** of **HypoKalemia**:

- Patients with hypokalemia do not always manifest ECG changes. In fact, the ECG of patients with even moderate hypokalemia may at times be *relatively* normal (*with little more than nonspecific abnormalities*).
- In contrast — Some patients who have *normal* serum K⁺ levels do manifest ECG changes that seem to suggest they have hypokalemia.
- **BOTTOM Line:** The ECG is simply not very accurate as a tool for assessing either the presence or severity of hypokalemia. This is not to say that the ECG should not be monitored in patients with hypokalemia. On the contrary — **ECG monitoring** may be **helpful** in a number of ways. These include: **i)** Detection of **arrhythmias** (*PVCs are common with hypokalemia; risk of VT is increased*); **ii)** The **QT (or QU) interval** may be significantly **prolonged** with hypokalemia — with associated *increased* risk of Torsades de Pointes (*Section 06.3*); and **iii)** *Serial* ECG changes that are followed as hypokalemia is treated and serum K⁺ returns to normal may provide insight to the likelihood of **associated ischemia** — vs ECG abnormalities being *primarily* a result of the electrolyte disorder. *Clinical correlation is KEY.*

FIGURE 11.7-1: What are the ECG Changes? Having conveyed potential clinical caveats of depending on the ECG to assess hypokalemia — We review in *schematic* Figure 11.7-1 those **ECG Changes** that *have* been associated with low serum K⁺ values:

- **Panel A** — The ST-T wave is normal. This is the *baseline* tracing.
- **Panel B** — shows relative *flattening* of the T wave and ST segment. This is typically the *earliest* change.
- **Panel C and Panel D** — In association with ST-T wave flattening (*and often with some degree of ST depression*) — a **U wave** develops (*Section 11.9*). A “pseudo-P-pulmonale” pattern (*with P wave peaking in inferior leads*) may sometimes be seen.
- **Panel E and Panel F** — ST depression becomes more noticeable and the U wave increases in size (*red arrow in Panel E*) — until ultimately the U wave *overtakes* the T wave. At this point, distinguishing between the T wave and U wave may be almost impossible (*ie, there is really “Q-U” rather than “Q-T” prolongation — as in Panel F*).

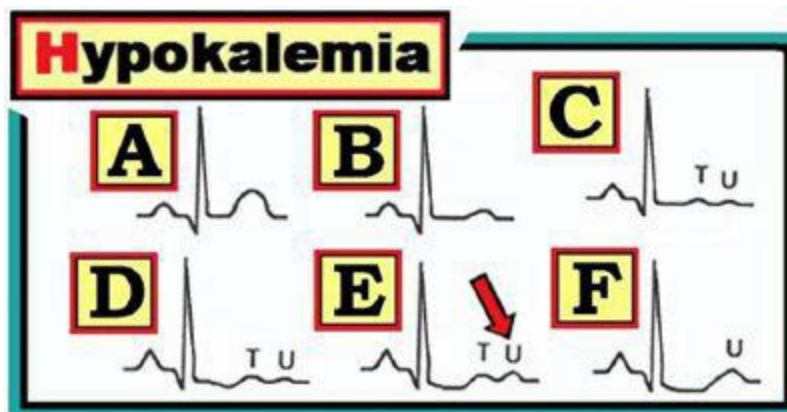


Figure 11.7-1: ECG Manifestations of *Hypokalemia*. Starting from a normal ST-T wave (**Panel A**) — the *earliest* change is ST-T wave flattening (**Panel B**). This may be followed by some degree of ST depression and development of a **U wave** (in **Panel C** and **Panel D**). ST depression becomes more noticeable and the U wave increases in size (*red arrow in Panel E*) — until ultimately the U wave *overtakes* the T wave. At this point, distinguishing between the T wave and U wave may no longer be possible (**Panel F**). Despite this well described *sequential* evolution of ECG changes — many patients with hypokalemia “fail to read the textbook” (See text).

11.8 – HYPOMAGNESEMIA: *Clinical Features/ ECG Signs*

In our introduction to this segment on electrolyte abnormalities — We referred to **magnesium** as the “*all-too-often forgotten cation*”. Serum Mg^{++} is still often *excluded* from routine chemistry profiles in many institutions. As a result — **Hypomagnesemia** often goes *undetected unless* the clinician is aware of the need to *specifically* assess for this cation.

- Hypomagnesemia is common in clinical practice. As is the case for potassium — the vast majority (>98%) of body magnesium resides *within* the intracellular compartment. As a result — serum levels (*measuring a minority of body magnesium that resides within the extracellular compartment*) do not reliably reflect body (and intracardiac) magnesium stores. Thus, it is not uncommon for a patient to have a serum Mg^{++} level that is still within the *low-normal* range (ie, *1.8 mg/dL*) — yet nevertheless having significant *body* depletion of this critical cation. **Therefore:** *Decidedly* low serum levels of Mg^{++} are diagnostic for hypomagnesemia — but blood levels in the *low-normal* range still *could* reflect low *body* stores that may nevertheless be clinically important.
- **Certain conditions predispose** to **Hypomagnesemia**. These include: **i)** associated electrolyte deficiency (*hypokalemia; hyponatremia; hypocalcemia; hypophosphatemia*); **ii)** Use of certain medications (*diuretics, digitalis*); **iii)** *Overuse* of Alcohol; **iv)** Diabetes and other metabolic disorders; **v)** renal disease; and **vi)** Certain *cardiac* conditions (*low Mg^{++} is common with acute MI; post-cardiac arrest; and in patients with various cardiac arrhythmias*). **KEY Clinical Point:** Consider hypomagnesemia (even if the blood level is within the low normal range) — in patients with any of the above potential *predisposing* conditions.
- **Hypomagnesemia** — may produce virtually *identical* ECG changes as may be seen with **hypokalemia** (Figure 11.7-1). That is — the ECG of a patient with low serum Mg^{++} may *vary* from showing little more than minimal nonspecific changes — to showing *diffuse* ST-T

flattening, ST depression, U waves, and *moderate-to-marked* QT (or QU) prolongation.

- Clinically — **Hypomagnesemia** often *coexists* with **hypokalemia**. The importance of *not* overlooking *coexistent* hypomagnesemia is that hypokalemia may prove *resistant* to K⁺ replacement until serum Mg⁺⁺ levels are restored to normal.

11.9 – U Waves: *Definition/Clinical Significance*

The most commonly cited ECG sign of hypokalemia is the presence of U waves. A **U wave** is recognized as the ECG deflection that occurs *after* the T wave — but *before* the next P wave (*red arrow in **Panel B** of schematic **Figure 11.9-1***). Physiologically — U waves are thought to represent repolarization of Purkinje fibers. We highlight the following clinical points about ECG recognition and the *clinical significance* of U waves:

- U waves are *not* always present on an ECG tracing (**Panel A** in *Figure 11.9-1*). When seen — they are often evident only in certain leads (*usually most easily recognized in leads II; V2, V3, V4*).
- U waves are not specific for **hypokalemia**. They may *also* be seen with **hypomagnesemia** — **LVH** — **bradycardia** — or simply on *normal* tracings *without* other abnormality.
- Like T waves — **U waves** may become *inverted* as a sign of ischemia (**Panel C**). This is usually quite subtle and *difficult* to see. To complicate recognition further — there may be ST depression with a *biphasic* terminal T wave (**Panel D**). As a result — **We Suggest** the following: IF not obvious on the patient's ECG — *Do not worry about U waves!*

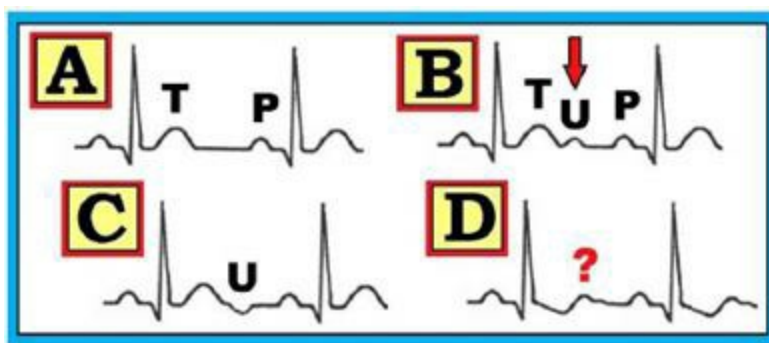


Figure 11.9-1: Recognition of U Waves on ECG. **Panel A** — A U wave will not always be seen on ECG. When present, a **U wave** is recognized as the ECG deflection that occurs *after* the T wave — but *before* the next P wave (*red arrow in **Panel B***). U waves are usually best seen in leads II; V2, V3, V4 — but they may be seen in any lead. They are not specific for hypokalemia (*U waves may also be seen with hypomagnesemia, LVH, bradycardia — and sometimes as a normal variant in healthy subjects*). **Panel C** — U waves may be subtle in appearance and they may be inverted (*sometimes as a sign of ischemia*). **Panel D** — It is *not* always easy to know if late baseline deflections reflect U waves or not (*See text*).

11.10 – **Figure 11.10-1: Electrolyte Disturbance or Ischemia?**

We conclude this segment on hypokalemia with the ECG shown below in **Figure 11.10-1**. This tracing was obtained from a patient with a history of alcohol abuse and *atypical* chest pain. *Obvious*

ST-T wave abnormalities are present.

- Given this clinical scenario — Are the ECG abnormalities seen likely to reflect *ischemia* or *electrolyte* disturbance? *How can you tell?*

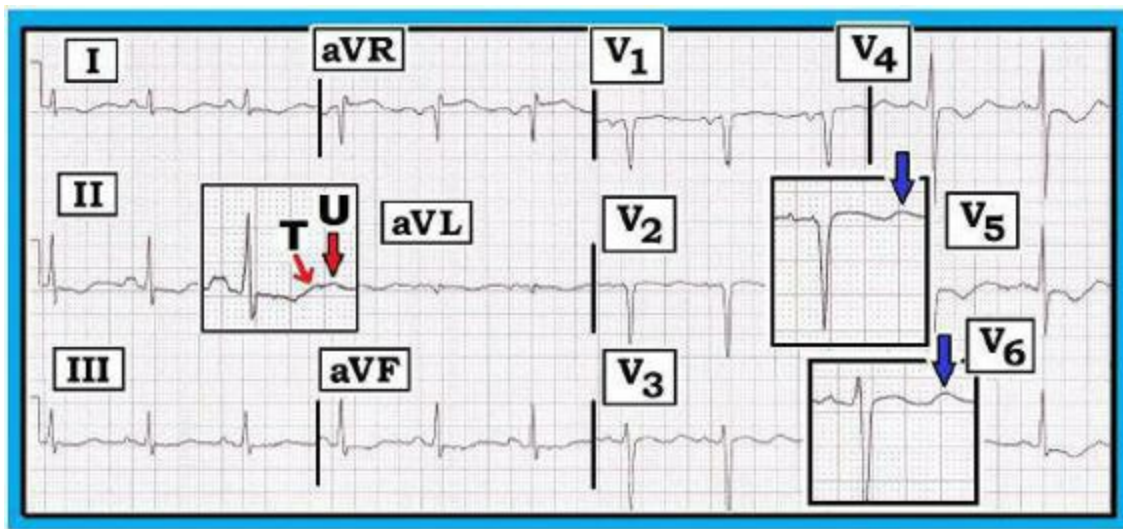


Figure 11.10-1: ECG obtained from a patient with a history of alcohol abuse and *atypical* chest pain. Are the ECG abnormalities seen likely to reflect *ischemia* or *electrolyte* disturbance? (See text).

Answer to Figure 11.10-1: There is sinus arrhythmia. PR and QRS intervals are normal — but the QT is **prolonged** (*clearly more than half the R-R interval in lead II, as well as in other leads*). The axis is normal. No chamber enlargement.

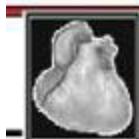
- The most remarkable finding is diffuse **ST-T wave flattening/depression** with **symmetric T inversion** in leads V4,V5,V6. In addition — **U waves** are seen in multiple leads (*most obvious in leads V2,V3 = blue arrows*). The U wave in lead II (*large red arrow*) seems to *blend* with the T wave in this lead (*similar to **Panel F** in Figure 11.7-1*).

Clinical Impression: There are 2 principle abnormalities in Figure 11.10-1: **i)** A *long* QT interval; and ii) *Diffuse* ST-T wave flattening, some ST depression, and *symmetric* T wave inversion. **Clinical correlation** is needed for optimal determination of *likely* etiologies. Comparison with a **prior** ECG (*if available*) — would be extremely helpful in knowing whether the changes seen are likely to be new or old.

- The **long QT interval** should prompt consideration of our **LIST #3** (*Section 06.2*). In brief — Consider: **i)** Drugs; **ii)** “Lytes” (*especially low K⁺ and/or low Mg⁺⁺ given this patient’s history of alcohol abuse*); and iii) CNS disturbance.
- Diffuse ST-T wave abnormalities** including ST flattening, ST depression and T wave inversion — should prompt consideration of our **LIST #4** (*Section 09.26*). Among potentially relevant entities to consider on this list in view of this patient’s history of alcohol abuse and chest pain are: **i)** drug effect (*if this patient was on digitalis or a diuretic*); **ii)** electrolyte disturbance (*especially low K⁺ and/or low Mg⁺⁺*); and iii) ischemia.

BOTTOM Line: There is *no way* to be certain from this *single* ECG whether the abnormalities seen in Figure 11.10-1 represent ischemia — electrolyte disturbance — or some combination of these, perhaps in association with other factors.

- **Clinical correlation** including lab values, serial tracings and close follow-up of this patient's clinical course — will be needed in order to know for certain. Even IF serum electrolyte values initially come back low — this would not exclude the possibility of *ischemic* T wave inversion *superimposed* on *electrolyte-induced* ST-T wave abnormalities.
- That said — We **strongly suspect hypokalemia** (*and probably also hypomagnesemia*) are at least *in part* responsible for the ECG abnormalities seen in Figure 11.10-1 because: **i)** the history of alcohol abuse predisposes the patient to low K⁺/low Mg⁺⁺ levels; and **ii)** the presence of very **prominent U waves** in several leads plus **marked QT prolongation** are both highly characteristic of these electrolyte disorders. Resolution of ECG abnormalities on serial tracings as electrolyte disturbance is corrected would confirm a *cause-and-effect* relationship.



Acute Pericarditis

Pericarditis is an inflammation of the fibrous lining of the heart. The **pericardium** itself is a *double-layered* membrane which covers the heart, as well as containing the roots of the great vessels. There are **2 layers** to the **pericardial lining**. These are: **i)** the inner **visceral pericardium** (*which is attached to the epicardium, or outer layer of the heart*); and **ii)** the **parietal pericardium** (*which is the outer layer of the pericardial sac*).

- Normally there is a potential *space* between the visceral and parietal pericardial layers = the pericardial “cavity”. Lubrication by a *small* amount of pericardial fluid allows *normal* free movement of the heart within the pericardial sac.
- Problems arise when because of inflammation (*or other insult to the pericardial lining*) — the amount of pericardial fluid increases. This results in pericardial “effusion. In its extreme — there may be pericardial *tamponade*, with inability of the heart to contract within the pericardial sac due to massive effusion *and/or* rigidity of the sac itself.

Clinical Points: The finding on **Echo** of a *small* “pericardial effusion” — is usually a normal phenomenon. Some fluid is needed for normal lubrication between visceral and parietal layers.

- **Echo** is an **excellent modality** for detecting pericardial fluid and estimating the amount of fluid. Anything more than a “small effusion” is *not* normal.
- Echo is invaluable as a bedside diagnostic aid for detecting larger effusions and determining IF pericardial tamponade is present.
- Simple viral pericarditis (*the most common kind of acute pericarditis*) — often does not produce any abnormality on Echo because: **i)** The pericardium is a thin layer. Inflammation generally produces *no* abnormality detectable on echo; and **ii)** Uncomplicated viral pericarditis usually does *not* produce significant pericardial effusion. **Therefore** — A *normal* Echo does not rule out *acute* pericarditis. On the contrary — a “normal” Echo is the most common finding you will see.

12.1 – Acute Pericarditis: How to Make the Diagnosis?

Pericarditis can be a *challenging* diagnosis to make. Recognition of **acute pericarditis** may be facilitated by thinking of the diagnosis as a 3-part process. These **3 components** consist of: **i)** History; **ii)** Physical examination; and **iii)** ECG findings.

History: The 1st step in making the diagnosis of *acute* pericarditis rests with obtaining the history. Information regarding the patient’s *prior* medical history — *recent* health issues — and the presence and nature of **symptoms** are all relevant.

- **Potential Etiologies** of *acute* pericarditis are *many* and extend *beyond* the scope of this ECG-

2014-ePub. The most common scenario is acute or recent **viral illness** occurring in a relatively *young* adult. Almost any viral agent may be responsible. That said — identification of the specific causative agent (ie, *by obtaining acute viral studies*) is often not undertaken because most cases of **acute viral pericarditis** (also known as “**idiopathic**” pericarditis) — are relatively benign, *spontaneously* resolve and do *not* recur. Knowing which virus is the “culprit” agent does not alter treatment, is expensive, and does not affect the ultimate course in most patients. **Clinical NOTE:** Given that **up to 90%** of all cases of acute pericarditis occurring in out-patients are viral (*idiopathic*) and usually occurring in *otherwise* healthy adults — **KEY Questions** to ask relate to: **i)** Whether there is a history of *preceding* viral illness; **ii)** *Prior* health status; and **iii)** *Previous* episodes (*viral pericarditis may recur, though this is not common*).

- **KEY Point** A **different** set of **diagnostic considerations** should be contemplated when considering acute pericarditis in an **older patient** with **underlying medical disorders**. Among the many possible *other* etiologies for pericarditis include: **i)** *Non-viral* infections (*tuberculosis, bacterial or fungal septicemia; immunocompromise*); **ii)** Uremia (*pericarditis is a common complication of end-stage renal disease*) ; **iii)** Malignancy (*metastasis to the pericardium*) ; **iv)** Collagen vascular disease (*rheumatoid arthritis; lupus; scleroderma*) ; **v)** *Post-radiation* pericarditis; **vi)** Chest trauma; **vii)** Post-myocardial infarction pericarditis (*Dressler syndrome*) ; **viii)** *Post-pericardiotomy* syndrome; and **ix)** *Still other* causes. **Clinical NOTE:** Pericarditis caused by *any* of the above entities is usually a **different disease** than uncomplicated *viral* pericarditis that occurs in a previously healthy young adult. The course is often far more severe, longer lasting, and much more likely to be associated with pericardial effusion *and/or* tamponade. Awareness of the patient’s **past medical history** may be **KEY** to suspecting the diagnosis for these other forms of *non-viral* pericarditis.

Symptoms: We *limit* discussion of symptoms to **Chest Discomfort**. Fever and dyspnea may also be seen with *acute* pericarditis — but these symptoms are nonspecific. The **chest pain** associated with **acute pericarditis** may be moderate or severe. It is characterized by being: **i)** *Pleuritic*; and **ii)** *Positional* in nature.

- **Pleuritic chest pain** — is often sharp in nature and increases with inspiration (*a result of associated pleural inflammation*).
- **Positional chest pain** — is typically relieved by sitting up and exacerbated by lying supine (*lying supine stretches the inflamed pericardium — whereas sitting up takes tension off the inflamed pericardium*).
- **Clinical NOTE #1:** Although the nature and severity of chest pain associated with *acute* pericarditis is highly variable — eliciting a **pleuritic** and/or **positional component** can be very helpful in suspecting the diagnosis, especially when the patient is a previously healthy young adult.
- **Clinical NOTE #2:** Given the common diagnostic dilemma of distinguishing between **ERP** (*Early Repolarization Pattern*) vs Pericarditis (*Section 12.9*) — it is helpful to remember that **acute viral pericarditis** is very **unlikely** in the **absence** of **symptoms**. In contrast — chest pain is *less* uniformly present in some of the other *non-viral* etiologies of *acute* pericarditis.

Physical Examination: There are **2 Goals** of physical exam: **i)** To try to make a *definitive* diagnosis by hearing a **pericardial friction rub**; **and** **ii)** to **rule out** **other** causes of chest discomfort (*pulmonary problems; musculoskeletal pain; herpes zoster; etc.*).

- Characteristics of a pericardial *friction* rub are that it is a **scratchy, superficial sound** (*like walking on snow*). There may be only one or several components to the rub. It may wax and wane in loudness — often becoming louder during inspiration. The rub is often transient, and may be intermittent — so it is worthwhile auscultating the patient on *at least* several occasions. **BOTTOM Line:** IF a pericardial friction rub is heard — then the diagnosis of acute pericarditis is made! However — *not* hearing a rub does *not* rule out pericarditis. **Clinical Reality:** Despite a cited incidence in the literature of well *over* 50% of patients with *acute* pericarditis having a rub at some point during their course — the diagnosis of many (*if not most*) cases of *acute* pericarditis is made in the *absence* of hearing a rub.
- As noted above — the history for *acute* pericarditis is often nonspecific (ie, *recent nonspecific viral infection*) — **and** the nature of chest pain may be variable. As a result — *other* causes of chest pain may be confused with the diagnosis. The other major **purpose** of **physical examination** is therefore to **rule out** some of these **other** pulmonary or musculoskeletal **causes** of chest pain.

12.2 – ECG FINDINGS of *Acute Pericarditis*

In clinical practice — the diagnosis of *acute* pericarditis is most often made on the basis of ECG findings. **ECG Changes** are divided into **4 stages**. The easiest way to remember these *sequential* ECG findings is to conceptualize the 4 stages as follows (**Figure 12.2-1**):

- **Stage I** — *Everything is UP*. That is — there is ST elevation in *almost* all leads (*except perhaps in the right-sided leads = leads III, aVR, V1*).
- **Stage II** — *Transition*. There may be “*pseudonormalization*” in this stage that temporally occurs *between* the diffuse ST elevation of Stage I **and** the diffuse T wave inversion to follow.
- **Stage III** — *Everything is DOWN*. That is — there is *diffuse* T wave inversion in virtually all leads.
- **Stage IV** — Normalization (*as the ECG changes of acute pericarditis eventually resolve*).

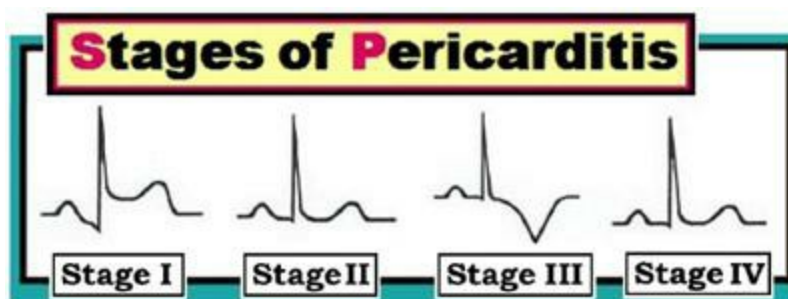


Figure 12.2-1: The 4 *Sequential Stages* of Pericarditis. **Stage I** (*diffuse ST elevation*) — is the *only* one of the 4 stages that is **diagnostic** of *acute pericarditis*. The amount of time spent in each stage is

variable. “Pseudonormalization” in **Stage II** — explains why even *relatively* new-onset pericarditis may sometimes manifest minimal ECG changes (*or no ECG changes at all*). The **Stage III** phase (*diffuse T wave inversion*) — is *nonspecific* and may be impossible to distinguish from ischemia. Ultimately, ECG changes resolve (**Stage IV**) — though this may take weeks or months. **PEARL:** To remember the 4 stages — Think *everything* UP (*1st Stage*) — transition (*2nd Stage*) — *everything* DOWN (*3rd Stage*) — Resolution (*4th Stage*).

KEY Points regarding Figure 12.2-1: The *amount* of **time** for *evolution* through the 4 ECG stages of acute pericarditis is **highly variable** (*it may take hours — to days — to weeks or more*).

- *Practically* speaking — the only ECG stage that is *diagnostic* is **Stage I**. This means that IF Stage I has *already* passed — it will *no longer be possible* to diagnose pericarditis by ECG (*because the ECG may almost look “normal” in Stage II — and in Stage III it shows diffuse T wave inversion that may be indistinguishable from ischemia*).
- Awareness of the above *expected* sequence of changes explains why the ECG may look relatively “normal” at an *early* stage in the process (ie, *during transitional Stage II in which ST segments return to baseline before going on to evolve into diffuse T wave inversion*).

Clinical Notes: Additional lab tests (*including Echo*) will often be ordered on patients with *suspected* pericarditis. That said — it is *rare* that these other tests will be “the test” that makes the diagnosis.

- **Echo** is *usually normal* — because the inflamed but nevertheless *thin* pericardial lining is *not* picked up by simple Echo unless an associated *significant* pericardial effusion is present (*Section 12.0*). Pericardial effusion is *absent* in many (*if not most*) cases of acute *viral* pericarditis that occurs in otherwise healthy subjects *without* other underlying systemic disease.
- The diagnosis of *acute* pericarditis can be *easy* IF — a **pericardial friction rub** is heard. But IF no rub is heard (*as is often the case*) — then **diagnosis** must be **presumptive** (*based on history and ECG findings*). Fortunately — a *presumptive* approach is reasonable given the generally *benign and self-limiting* course of most cases of *acute viral (idiopathic)* pericarditis that occur in otherwise healthy individuals.

12.3 – Stage I of Acute Pericarditis

Given that **ECG diagnosis** of *acute pericarditis* can really only be **made** in **Stage I** — We focus on recognizing ECG findings in this stage (**Figure 12.3-1**):

- There will *often* be **sinus tachycardia**. While sinus tachycardia is a *nonspecific* finding that is *not* essential for diagnosis of *acute pericarditis* — the *absence* of at least a *relatively* rapid heart rate is a factor against the diagnosis of *acute pericarditis*.
- There is **diffuse ST segment elevation** (*Stage I is the “everything UP” stage*). This generalized ST elevation is thought to reflect a diffuse *subepicardial* injury current. The only leads that do *not* consistently show ST elevation are one or more of the *right-sided* leads (*III*,

aVR and *V1*) that view the heart from a more distant perspective (*leads shaded in blue in Figure 12.3-1*).

- The **shape** of **ST elevation** with acute pericarditis is often quite *similar* to that seen with early repolarization (**concave up** = “smiley” configuration, sometimes with J-point notching). It is almost as if the ST segment itself is normal and has been “lifted” above the baseline.
- With *acute* pericarditis — the appearance of **ST segments** in **leads I and II** tends to look **similar**. This differs from the usual situation with acute *inferior* MI — in which lead III is much more likely to resemble lead II.
- There is **no reciprocal ST depression**! This is a key distinguishing feature of acute pericarditis from *acute* MI.
- *Infarction* Q waves are absent (*No more than small q waves are seen*).
- **PR segment depression** is often noted in **several leads** (*seen in Figure 12.3-1 in leads I, II and V2, V3 — but not in leads aVL, aVF; V4, V5, V6*). PR depression is thought to reflect an atrial injury current. As was the case with atrial infarction (*Section 09.35*) — look to **lead aVR** for **reciprocal PR elevation** (*which is present in Figure 12.3-1*).

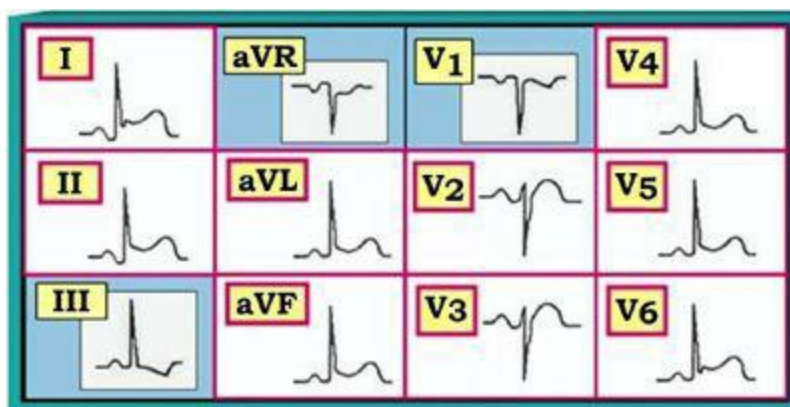


Figure 12.3-1: ECG Findings in **Stage I Acute Pericarditis**. The most characteristic finding is **diffuse ST elevation** in all but one or more of the “right-sided” leads (*leads III, aVR, V1*). ST elevation is generally concave up (“smiley”-shape) — although *coved* ST elevation is seen here in leads V2, V3. As commonly occurs with early repolarization — there may be J-point notching in one or more leads (*seen here in leads I, V6*). Lead I often resembles lead II (*rather than lead III, as would occur with acute inferior MI*). There is *no* reciprocal ST depression. Finally, **PR depression** is often seen in at least several leads (*here in leads I, II and V2, V3*) — with PR elevation in lead aVR.

12.4 – PR Depression: How Helpful a Sign is this?

Normally — the **PR segment** is *isoelectric* with respect to the ST segment baseline (**Panel A** in *Figure 12.4-1*). It is from this PR segment baseline that we generally judge ST segment deviations (*elevation or depression*) — assuming baseline wander is minimal and that the heart rate is not so fast as to preclude identification of the PR segment (*Section 09.15*).

- With generalized inflammation of the pericardial lining — **depression** of the **PR segment** below the ST baseline will often be seen in *at least* several leads. Recognition of **PR depression** should be evident in **Figure 12.4-1** by comparing the PR segment in **Panel B** (*relative to its*

dotted ST segment baseline) — with the normal (*isoelectric*) PR segment seen in Panel A.

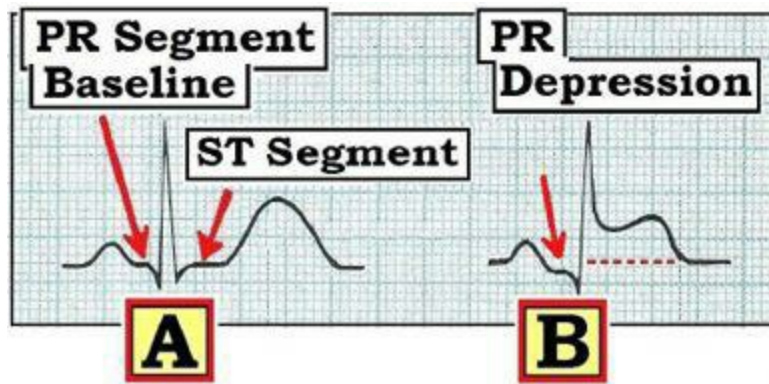


Figure 12.4-1: Comparison of a *normal* PR segment (Panel A) — with PR depression seen in Panel B, in which the PR segment descends *below* the ST baseline (*See text*).

Clinical CAVEATS: PR depression is often subtle. As a result — this finding is subject to *inter- and intra-observer* variability.

- PR depression is *not* always seen with *acute* pericarditis.
- PR depression may occasionally be seen in *other* conditions. These include: **i)** normal repolarization variants; **ii)** patients with acute MI (*and/or with atrial infarction*); **and iii)** with tachycardia (*the PR shortens with tachycardia, which makes PR assessment more challenging at faster heart rates*).

BOTTOM Line: Be aware of the term **PR depression** — as this will often come up in *diagnostic* discussion about possible *acute* pericarditis. Definite identification of PR depression in *several* leads is possible — and is diagnostically useful (**Figure 12.4-2**). Support that the finding of PR depression is *real* and meaningful may be derived by the finding of **PR elevation** in lead **aVR**. That said — Be aware that *both* false *positive* and false *negative* diagnosis of acute pericarditis is frequently associated with the phenomenon of seeing PR depression.

- **Therefore** — It is probably best *not* to base your diagnosis of *acute* pericarditis on whether or not you see PR depression.

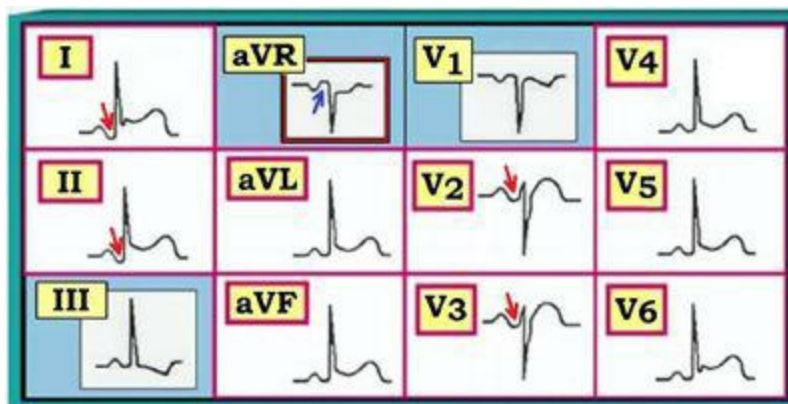


Figure 12.4-2: Schematic tracing illustrating ECG findings of *acute pericarditis* (reproduced from Figure 12.3-1). In addition to seeing *diffuse* ST elevation in the *absence* of reciprocal ST depression — **PR depression** is seen in several leads (*red arrows*). Support that this finding of PR depression is real — is forthcoming from the finding of **PR elevation** in lead **aVR** (*blue arrow*). While not enough by itself to make the diagnosis of acute pericarditis — PR depression (*in association with PR elevation in lead aVR*) is clearly supportive of the diagnosis (*See text*).

12.5 – What is *Spodick’s Sign*?

An additional ECG diagnostic sign that is often mentioned with discussion of *acute pericarditis* — is ***Spodick’s sign*** (named after Dr. David Spodick who is internationally known for his work on pericarditis).

- ***Spodick’s Sign*** — is a *downsloping* of the **TP (or entire QRS-TP) segment** that may be present in a number of leads with *acute pericarditis* (*downward slanting red dotted line in Figure 12.5-1*). When seen in at least a few leads in association with a *suggestive* clinical history and *other* typical ECG findings — this sign further supports the diagnosis of *acute pericarditis*.

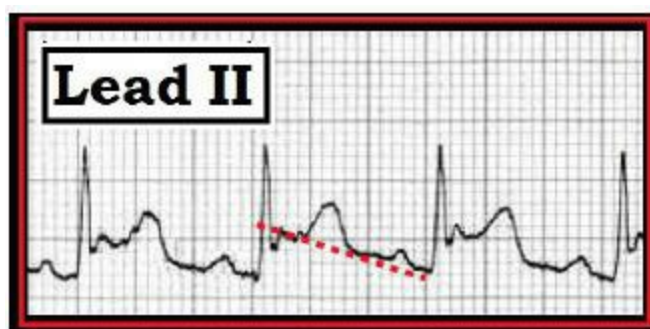


Figure 12.5-1: Spodick’s sign — defined as a *downsloping* of the TP (or entire QRS-TP) segment (*red dotted line*). ***Clinical Caveat*** — In our experience, determination of whether or not ***Spodick’s sign*** is present is often *subjective* and *suboptimally* reproducible. While we regularly look for this sign when contemplating the diagnosis of acute pericarditis — We caution against depending on presence or absence of Spodick’s sign as your sole criterion for diagnosing acute pericarditis (*See text*).

12.6 – *Differential Diagnosis: Acute MI vs Early Repolarization?*

Distinction between Stage 1 pericarditis and early repolarization or acute MI can *usually* be made because:

- ***Early Repolarization*** — is most often seen in otherwise *healthy* young adults. ST elevation is usually *localized* to one (*at most two*) areas of the heart — vs **more *diffuse* ST elevation** with *pericarditis*. Be aware that the *shape* of ST segment elevation may be similar with both conditions (“*smiley*” configuration) — and J-point notching may be seen in both conditions.
- ***Acute MI*** — is usually suggested by the history (*older patient with risk factors; chest pain is more constant and severe* — and *chest pain is less likely to be pleuritic or positional*). The ECG may show **Q waves** — and Q waves may *increase* in size as the infarct evolves. There

will usually be at least some **reciprocal ST depression** during the course of *acute MI* — whereas neither pericarditis nor early repolarization manifest *reciprocal* changes.

PEARL: A distinguishing feature between acute MI and pericarditis — is that with *acute MI*, T wave inversion will often be seen while the ST segment is *still* elevated (**Figure 12.6-1**). In contrast, with *acute pericarditis* — ST segments are first diffusely elevated (*1st Stage*) — then ST segments return to baseline (*2nd Stage*) — and only then is there T wave inversion (*3rd Stage*). Thus, the picture in *schematic Figure 12.6-1* is strongly suggestive of **acute MI** rather than pericarditis because the T wave is inverted while the ST segment is still elevated.

- **Final Point:** As previously noted (*in Section 12.3*) — ST segments in leads I and II tend to look similar with *acute pericarditis*. This is in contrast to what is usually seen with *acute inferior MI* — in which case lead III is much more likely to resemble lead II in appearance.



Figure 12.6-1: With *acute Pericarditis* — the T wave typically does not invert until after the ST segment has returned to baseline (*Section 12.2*). In contrast, with **acute MI** (*as seen here*) — the T wave may invert while the ST segment is *still* elevated.

12.7 – Acute Myocarditis/Endocarditis: ECG Changes?

The causes and symptoms of **acute Myocarditis** and **Endocarditis** are *many* and extend *beyond* the scope of this ECG PB book. Our purpose in this brief subsection is merely to increase awareness of the ECG abnormalities that *may* be seen. The ECG picture is not **predictable** with either *myocarditis* or *endocarditis*. It may include:

- Various forms of SA and AV nodal block (*from 1st degree — up to 3rd degree AV block with long pauses*).
- Conduction system defects (*BBB, hemiblocks, IVCD*).
- Atrial and ventricular arrhythmias (*including VT/VFib*).
- The *gamut* of ST-T wave abnormalities (*ST elevation/depression; T wave inversion; nonspecific ST changes*).

BOTTOM Line: ECG findings with myocarditis and endocarditis are highly variable and nonspecific. Abnormalities may be minimal or marked. Awareness of this variability may help to explain *extreme* abnormalities that may be seen when *either* myocarditis or endocarditis are suspected.

12.8 – FIGURE 12.8-1: *Acute MI or Pericarditis?*

Consider the ECG shown in Figure 12.8-1 — obtained from a 35-year old man with *atypical* chest pain.

- Are the findings suggestive of *acute MI* or *acute pericarditis*?
- *How certain* are you of your diagnosis?

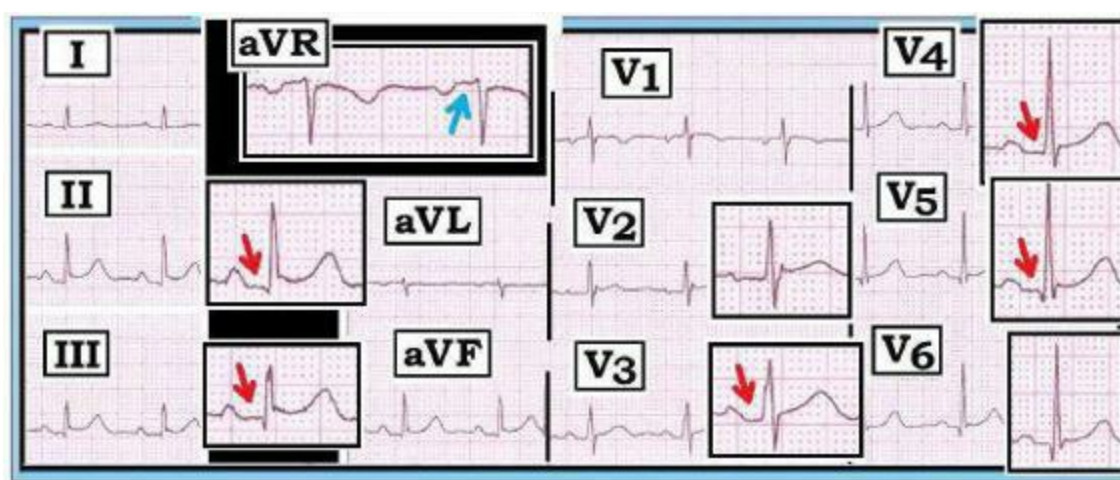


Figure 12.8-1: *Acute MI vs Acute Pericarditis.*

Answer to Figure 12.8-1: The rhythm is sinus. All intervals and the axis are normal. No chamber enlargement. Early transition (*the R wave becomes taller than the S wave is deep by lead V2*). Otherwise — the principal finding is **diffuse ST elevation** that is present in virtually all leads *except* for leads I, aVR and aVL.

- The amount and extent of ST elevation in Figure 12.8-1 is *more* than is usually expected with early repolarization. ST elevation is concave up. There is J-point notching or slurring in a number of leads.
- **PR depression** is seen (*red arrows*). In addition — there is **PR elevation** in lead aVR (*blue arrow*). Together with the presence of *diffuse* ST elevation — this all favors **acute pericarditis** as the diagnosis.
- **P.S.:** We find it *difficult* to tell if **Spodick's sign** (*Section 12.5*) is present or not in leads II and III of Figure 12.8-1. As previously noted — We caution *against* dependence on this sign when its presence is questionable because of subjectivity in its interpretation.
- **Finally** — The likelihood of *acute MI* is low. This is because: **i)** ST elevation is *diffuse* and manifests an upward concavity with J-Point notching; and **ii)** There is *no* reciprocal ST depression. In contrast — the ST elevation of *acute MI* is typically coved; localized to one (*or at most 2*) lead areas; not notched; and associated with *reciprocal* ST depression. The patient's

relatively *younger* age and *atypical* symptoms in this case are also *against* acute MI. This patient did have **acute pericarditis**.

12.9 – FIGURE 12.9-1: *Pericarditis or Early Repolarization?*

We conclude this segment on *acute* pericarditis by revisiting the ECG shown in Figure 12.9-1 — which we *initially* encountered in Section 09.19 when discussing early repolarization.

- How would you interpret this tracing IF told that it was obtained from an otherwise *healthy* young adult?
- Would your interpretation *change* — IF told that this young adult had a *recent* upper respiratory infection and was complaining of *pleuritic-type* chest pain?
- *Despite* ST elevation in *several* leads — Why is the ECG shown in Figure 12.9-1 not suggestive of an *acute* MI?

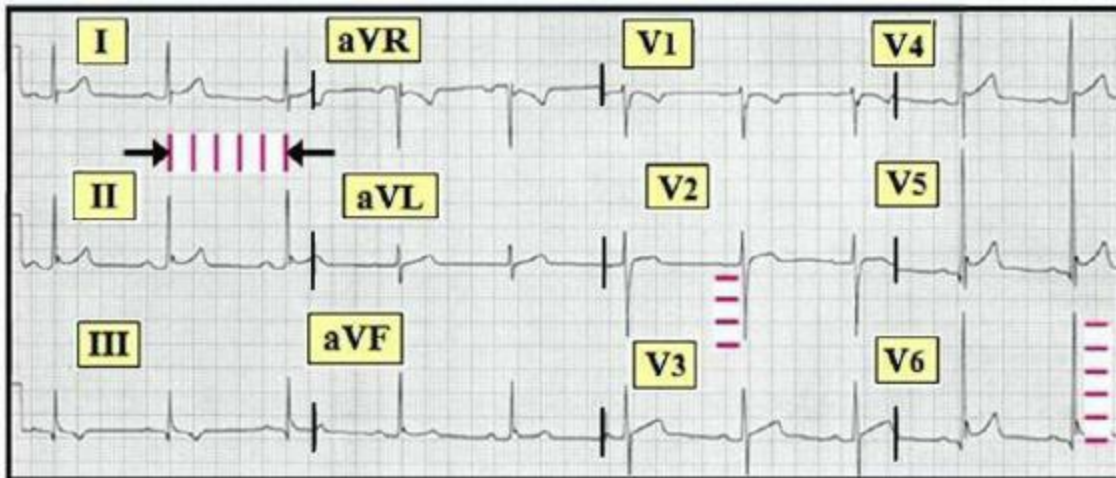


Figure 12.9-1: ECG obtained from a young adult (*reproduced from Figure 09.19-1*). Would your interpretation of this tracing *change* IF the clinical scenario was *recent* URI with *pleuritic* chest pain? — vs *sudden-onset* of *crushing* chest pain in an *older* patient? — vs a young adult who was *not* having symptoms? (See text).

Answer to Figure 12.9-1: As emphasized in Section 09.19 — our *descriptive* analysis for ECG findings on this 12-lead tracing does not change regardless of the history. Only the *relative* probabilities for our clinical impression will change depending on the clinical scenario.

- **Descriptive Analysis:** There is sinus rhythm with normal intervals and axis. Voltage for LVH would *not* be present if the patient was younger (*less than 35 years of age*). Transition is normal (*occurs between lead V3-to-V4*).
- A Q wave with T wave inversion is seen in lead III. Small, narrow q waves are seen in V5, V6. Taken in isolation — *none* of these findings are necessarily abnormal.
- Instead — the most remarkable finding on this ECG is **ST elevation** in **multiple** leads. This ST elevation manifests an *upward concavity* (“*smiley*”-configuration) in most leads in which it is seen — although the ST segment is *coved* in lead V2, and the ST segment takeoff is *straightened*

in lead V3.

- **J-point notching** is seen in *several* leads. This is most notable in leads V5,V6 — but *also* present in leads I,II,aVF and V4. The *shape* of this J-point notching is characteristic of early repolarization.
- *Infarction* Q waves are absent. That is — the q waves in leads V5,V6 are small and narrow, with the appearance of being normal *septal* q waves. The Q wave in lead III is deeper — but as an *isolated* finding, this is *not* necessarily abnormal (*Section 09.12*).
- There is *no* reciprocal ST depression (*isolated T wave inversion in lead III does not qualify in this case as “reciprocal” ST depression*).
- We do not appreciate PR depression.
- Lead II seems to resemble lead I more than lead III.
- We can easily “*talk ourself into*” a positive Spodick sign for the sloping TP segment in leads V4,V5,V6.

Putting Together ECG Findings in Figure 12.9-1: Our reason for presenting this ECG again (*reproduced from Figure 09.19-1*) — is to emphasize: **i)** How *challenging* it may sometimes be to assess the ECG of a patient with chest pain and ST elevation; and **ii)** How important the clinical scenario is in our interpretation.

- IF this patient was an ***asymptomatic*** otherwise healthy **young adult** — We would not hesitate calling this ***Early Repolarization*** (*concave up ST elevation in multiple leads; J-point notching; no infarction q waves and no reciprocal ST depression*).
- On the other hand — IF this young adult had ***pleuritic chest pain with*** a ***recent*** URI — We would have to be concerned about the *possibility* of ***acute pericarditis*** (*diffuse ST elevation and lack of reciprocal ST depression in the setting of a history consistent with acute pericarditis*). Although the *slow* heart rate and highly characteristic J-point notching seen here might lead us to question this diagnosis — in the absence of a prior tracing to document longstanding early repolarization, it would be difficult to exclude acute pericarditis.
- Finally — IF this patient was ***older with new-onset chest discomfort*** — All bets would be off (*especially in the absence of a prior ECG for comparison*)! Although the ECG in Figure 12.9-1 does not show infarction Q waves, *reciprocal* ST depression, or ST coving more suggestive of acute injury — the onus of proof when the history is concerning falls on us to *rule out* the possibility of early acute MI (*rather than having to rule it in*). At the least, we would start by *repeating* the ECG in short order to see if any evolution occurs. Whether or not to admit this patient (*and/or to consider acute investigation*) — would depend on a series of factors as discussed in detail in Section 10.

BOTTOM Line: An ECG such as that seen in Figure 12.9-1 — could be *consistent* with *acute pericarditis* or even early *acute* injury — IF clinical features suggesting either diagnosis were present. As relevant to Section 12 — Assessing the patient with chest pain for the possibility of *acute pericarditis* is often a *challenging* task that entails far *more* than simply looking at the patient’s ECG.

- **P.S.** — It turned out in this case that the ST segment elevation seen in *multiple* leads in Figure

12.9-1 was a **normal repolarization variant** (*and not due to acute coronary syndrome or pericarditis*).

- **PEARL:** Making this patient a ***miniaturized* copy** of his **ECG** that he can carry in his wallet may be a prudent way to *avoid* unnecessary hospital admission in the event chest discomfort is experienced in the future.

13.0 – Computerized ECG Interpretations



Computerized ECG Interpretations

A frequent question that arises is, “How best to use (*or not use*) the **computerized ECG interpretation**?” *Opinions vary*. We feel the answer depends on the goals and *experience* level of the interpreter.

- *Computerized* ECG analysis systems are **not infallible**. Although they clearly have merit in certain regards — they are far from perfect at ECG interpretation. Our task is to *appreciate the positives* of computer systems *while* being aware of their drawbacks.

13.1 – Computerized Systems: Pros & Cons

At the current time — virtually all modern ECG machines *automatically* provide a **computerized interpretation**. This has *benefits* and *drawbacks*. Consider the following:

- *Computerized* systems **excel** at computing **values**. This is because *that’s what computers do*. As a result — *computerized* systems are **extremely accurate** in calculating: **i)** Rate; **ii)** Intervals (*PR/QRS/QT intervals*); and **iii)** Axis.
- *Computerized* systems are *usually* reliable in recognizing **sinus rhythm** mechanisms and **normal tracings**.
- For the **Expert Interpreter** — the *best* feature of *computerized* systems is that they **save time!** There is *no longer need* to calculate rate, intervals or axis — since the computer *instantly* provides legible and *accurate* print-out of these values. IF the computer says, “*Normal ECG*” — it may literally take *no more* than 2-3 seconds for an *experienced* interpreter to peruse the tracing and *sign* the report (*provided there is agreement with the computer interpretation*).
- For the **Non-Expert Interpreter** — the major benefit of *computerized* systems is the **backup opinion** the system provides. The computer may suggest findings *not* initially thought of by a *less* experienced interpreter. This encourages more careful, targeted review of the tracing. It may also be *educational* by the suggestions it makes. Finally — confidence is boosted when computer analysis agrees with the clinician’s interpretation.

NOTE: The computer backup opinion **may also help** the **expert-in-a-hurry** by *reducing* the chance that any ECG findings will be overlooked.

- Interpretation of any one ECG by an expert provided with: **i)** a moment of time; and **ii)** the clinical history — will always be superior to interpretation by a machine. That said — this is not reality.
- Reality in the “real world” — is that the clinician assigned to interpret *all* tracings on a given hospital or ambulatory service usually has *limited* time to interpret a *large* number of ECGs and

is often asked to do so without the benefit of clinical history. As a result — it becomes *easy* for even an expert interpreter to *overlook* certain findings on occasional tracings. **Knowing how to use** the *computerized* interpretation as a “**backup opinion**” can be invaluable even for the most experienced of interpreters! (Grauer, Nelson, Marriott et al: *J Am Bd Fam Prac* 1:17-24, 1989).

CAVEATS (What the Computer May Miss): *Computerized* systems do not do nearly as well in evaluation of abnormal tracings as they do in assessing ECGs with minimal abnormalities. The more complex the abnormal ECG is — the more difficult it becomes for a *computerized* system to render an entirely accurate interpretation.

- *Computerized* systems are far less accurate interpreting rhythms that do *not* have a sinus mechanism.
- They may miss *subtle* infarctions.
- They tend to *overinterpret* the J-point ST elevation that is commonly seen with early repolarization patterns. As a result — *computerized* systems may be prone to mislabel these normal variants as “acute MI”.
- *Computerized* systems may *miss* pacemaker spikes/WPW/*tall* R in V1. They are *unlikely* to appreciate certain clinical entities such as Wellens’ syndrome or DeWinter T waves.
- Many hospitals do not utilize *special* computer programs for interpretation of ECGs obtained on *pediatric* patients. Obvious problems with interpretation will arise IF a *pediatric* ECG is interpreted by a computer program using *adult* criteria.
- Finally — *computerized* systems by definition lack the “*human Gestalt*” by an expert of the overall tracing.

13.2 – ***Suggested Approach: How to Use the Computer***

The *most* important point to emphasize in this Section — is that clinical use of the *computerized* report by *non-expert* interpreters should be very *different* than use of this same report by the *expert* who regularly interprets a large volume of tracings. ***Expertise of the interpreter*** therefore ***dictates the approach we recommend*** (Grauer: *Practical Guide to ECG Interpretation*; Mosby, St. Louis; pp 375-379, 1998).

- For the ***Non-Expert Interpreter*** — Do not initially read the computer report. Instead — ***WRITE OUT (or at least think out)*** your interpretation first. Only after independently making your own interpretation — should you look at the computerized report. At this point — Check *each* of the findings you note with *each* computer statement. Then delete, modify *and/or* add to the computer interpretation as needed.
- For the ***Expert Interpreter*** — Review the computer report *either* before or after evaluation of the ECG itself. *Minimize* time devoted to determination of heart rate, intervals and axis (*since the computer is very accurate for these parameters*). Consider more careful evaluation IF the rhythm is *not* sinus — or IF the ECG is interpreted by the computer as abnormal. Overread *each* computer statement. Place a ***check mark*** next to those that are accurate. Delete, modify or add to

incorrect statements.

KEY Point: The *expert interpreter* is *not* using the *computerized* report to “learn”. This is because by definition — the interpretation of an *expert electrocardiographer* is the “**gold standard**”. Since computerized systems are programmed by experts — the *best* they can realistically hope for is to put out interpretations that *equal* the level of accuracy of the expert that programmed them.

- The *expert* uses the computer: **i)** to save time; **and ii)** to prevent overlooking findings when forced to read *many* ECGs in a *limited* period of time.
- **Less experienced interpreters** do look to the computer to assist in accuracy. They are usually called on to read *no more* than one ECG at any one time. Therefore — the *most* important step for the *non-expert* is to first *COVER UP* the computerized report. It is otherwise all too easy to be biased by what the computer says. Used in this way — comparing one’s *own* interpretation with what the computer says *optimally* incorporates potential benefit from any discrepancy in interpretation that may exist.

13.3 – FIGURE 13.3-1: *Do You Agree with the Computer?*

Perhaps the *best* way to illustrate potential pros and cons of *computerized* interpretations — is by clinical example. Consider the ECG shown in **Figure 13.3-1** — obtained from a 78 year old woman with *atypical* chest pain.

- The **computerized interpretation** was: Sinus rhythm; left axis (*-10 degrees*) — but otherwise “normal” ECG.
- *Do you agree* with the *computerized* interpretation?
- **HINT:** Be sure to interpret this ECG *in its entirety* by the systematic approach first — *before* you compare what the computer said with your interpretation.

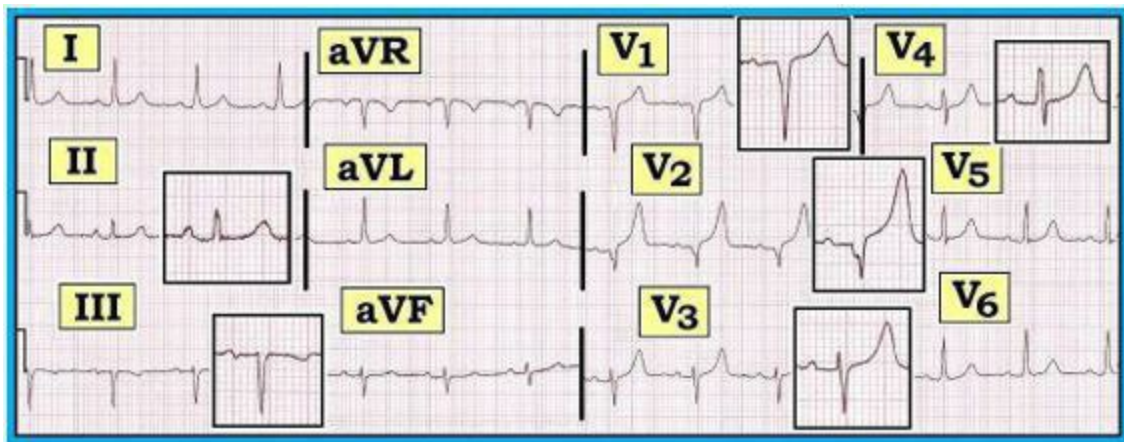


Figure 13.3-1: ECG obtained from a 78 year old woman with *atypical* chest pain. The **computerized report** interpreted this tracing as, “left axis but otherwise normal”. *Do you agree* with the *computerized* report?

Answer to Figure 13.3-1: The rhythm is sinus. All intervals are normal. The axis is leftward (*predominantly negative QRS in lead aVF*) — but *not* negative enough to qualify as LAHB (*since the QRS in lead II is still upright*). No chamber enlargement.

- Regarding **Q-R-S-T Changes** — There are **QS complexes** in leads V1,V2. An r wave develops by lead V3 — and transition occurs normally between lead V3-to-V4. Although there is *no more* than minimal (*at most*) ST elevation — **T waves** are **dramatically peaked** in anterior precordial leads (*especially in lead V2*). There is shallow T inversion in lead III, and perhaps some *nonspecific* ST-T wave flattening in lead aVF.

IMPRESSION: This example highlights the importance of *overreading* the *computerized* interpretation after you have *independently* arrived at your own conclusion. This is not a “normal” ECG. That statement should be ***crossed out*** on the computerized report. This is because the *computerized* interpretation is a medical record — and statements you disagree with should therefore be crossed out.

- **Clinical correlation** is needed to determine the meaning of the *abnormal* findings you identified. **Of Concern** — is the fact that **i)** this woman is of a “certain age” (78 years old — *so clearly old enough to have coronary disease*); — and **ii)** she is having “chest pain” (*even though it is described as “atypical” in nature*).
- While *not* definitive — the **QS complexes** in leads **V1,V2** could reflect *septal* infarction of *uncertain* age. This should *at least* be noted in your interpretation (*it was ignored by the computerized report*).
- There is **marked T wave peaking** — especially in **leads V2,V3**. This is not normal (*despite also being ignored by the computerized report*). Possible explanations for this *abnormal* T wave peaking include: **i)** Hyperkalemia (*less likely because T wave peaking is not generalized and the base of these T waves is not narrow — but a serum K+ level should nevertheless be checked to rule this out*); **ii)** Ischemia (*which when posterior in location sometimes manifests as anterior T wave peaking*); and **iii)** DeWinter T waves. Given the history of chest discomfort — we are *most* concerned with this 3rd possibility. While the J-point ST depression that is usually seen with **DeWinter T waves** is *missing* in Figure 13.3-1 — the ECG picture in this tracing is otherwise perfectly compatible with this harbinger sign of possible impending proximal LAD occlusion (*Section 10.58*).

BOTTOM Line: It might be easy to *overlook* the QS complexes in leads V1,V2 of this tracing IF you allowed the computerized report to *bias* you prior to rendering *your own* independent interpretation. Hopefully — you did *not* overlook the obviously abnormal T wave peaking in anterior leads that somehow escaped detection by the computer. Recognition of **DeWinter T waves** is indication for *immediate* cath/acute reperfusion — so this possibility mandates immediate attention. This would have been *missed* had the computer report been accepted without overread. *Computerized* interpretations can be extremely helpful to *both* expert and *non-expert* interpreters — but knowing *HOW* to use the computer report always assumes *first* priority.



We conclude this ECG-2014-ePub with brief comment on the fascinating phenomenon of **electrical alternans**. This relatively uncommon clinical entity is frequently misunderstood — and often overlooked when it does occur. Electrical alternans is a general term that encompasses a number of *different* pathophysiologic mechanisms. Its occurrence is *not* limited to pericardial tamponade — but instead has been associated with an expanding array of clinical conditions.

- Distinction should be made between *electrical* and *mechanical* alternans. The term “**alternans**” itself — merely indicates that there is **phasic fluctuation** in **some cardiac signal** from one beat to the next within the cardiac cycle. This may be in the strength of the pulse (*or the blood pressure recorded*) — or it may be in one or more waveforms in the ECG recording.

NOTE: Discussion is *limited* in this Section 14 to ECG manifestations of alternans. Nevertheless — it may be helpful to first define *other* alternans phenomena that may sometimes be confused with the various ECG manifestations (*especially since these other forms of alternans phenomena may also be seen with cardiac tamponade*).

- **Pulsus alternans** — is a *mechanical* form of alternans. The rhythm is *regular* — but cardiac output varies from beat-to-beat. It is seen with severe systolic dysfunction. Pulsus alternans should be distinguished from a **bigeminal pulse** — in which a *weaker* beat follows the stronger beat by a *shorter* time interval (*as occurs when the alternating beat is a PVC, which understandably generates less cardiac output*).
- Pulsus alternans should also be distinguished from **pulsus paradoxus** — in which there is a palpable decrease in pulse amplitude (*or a measured drop of >10mm in blood pressure*) during quiet inspiration. While *pulsus alternans* and *paradoxus* may both be seen with pericardial tamponade — they are *different* phenomena than the various types of *electrical* alternans.

14.1 – Electrical Alternans: *Definition/Features/Mechanisms*

Electrical alternans — is a *beat-to-beat* variation in any one or more parts of the ECG recording. It may occur with *every-other-beat* — or with some other recurring ratio (3:1; 4:1; *etc.*). Amplitude or direction of the P wave, QRS complex, ST segment *and/or* T wave may all be affected (*although P wave alternans is rare*). Alternating interval duration (*of PR, QRS or QT intervals*) may also be seen.

- **Electrical alternans** — was first observed in the laboratory by Herring in 1909. It was reported clinically by Sir Thomas Lewis a year later, who characterized the phenomena as occurring, “*either when the heart muscle is normal but the heart rate is very fast or when there is serious heart disease and the rate is normal*”. This 1910 description by Lewis serves well to this day to remind us of the 2 principal clinical situations in which electrical alternans is most

often encountered: **i)** Supraventricular *reentry* tachycardias; and **ii)** Pericardial tamponade.

Mechanisms: There are 3 basic types of electrical alternans phenomena — each relating to a different pathophysiologic mechanism: **i)** *Repolarization* alternans; **ii)** *Conduction* and *Refractoriness* alternans; and **iii)** Alternans due to abnormal cardiac motion. A common cellular mechanism may underlie each of these processes relating to abnormal calcium release or reuptake within the sarcoplasmic reticulum.

- ***Repolarization* alternans** — entails *beat-to-beat* variation in the **ST segment** *and/or* **T wave**. Alternation in ST segment appearance (*or in the amount of ST elevation or depression*) — is often linked to ischemia. In contrast — T wave alternation is more often associated with changes in heart rate or in QT duration (*especially when the QT is prolonged*). In patients with a long QT — T wave alternans may forebode impending Torsades de Pointes. Both ST segment and T wave alternans have been known to precede malignant ventricular arrhythmias. Thus, this type of electrical alternans may convey important adverse prognostic implications when seen in certain situations. That said — a *variety* of clinical conditions have been associated with ***repolarization* alternans**, such that adverse prognostic implications do not always follow. Among these clinical conditions are congenital long QT syndrome — severe electrolyte disturbance (*hypocalcemia; hypokalemia; hypomagnesemia*) — alcoholic or hypertrophic cardiomyopathy — acute pulmonary embolus — subarachnoid hemorrhage — cardiac arrest and the post-resuscitation period — and various forms of ischemia (*spontaneous or induced by treadmill testing or other stimulus*).
- ***Conduction* and *Refractoriness* alternans** — entails variance of impulse propagation along *some* part of the conduction system. This may result from fluctuations in heart rate or in nervous system activity or from pharmacologic treatment. ECG manifestations from this form of alternans may include ***alternating appearance*** of the **P wave**, **QRS complex** or alternating difference in **P-R** or **R-R interval duration**. In particular — QRS alternans during *narrow* SVT rhythms has been associated with ***reentry* tachycardias**. While identification of **QRS alternans** during a *regular* SVT often indicates *retrograde* conduction over an AP (*Accessory Pathway*) — this phenomenon has also been seen in patients with simple PSVT/AVNRT that exclusively limits its reentry pathway to the AV Node. Therefore — identification of QRS alternans during a *regular* SVT does not prove the existence of an accessory pathway. *Conduction* and *refractoriness* alternans may be seen with *WPW-related* as well as *AV Nodal-dependent* reentry tachycardias — atrial fibrillation — acute pulmonary embolus — myocardial contusion — and severe LV dysfunction.
- ***Cardiac Motion* alternans** — is the result of cardiac movement rather than electrical alternation. The most important clinical entity associated with ***motion* alternans** is large pericardial effusion — though motion alternans has also been observed in some cases of hypertrophic cardiomyopathy. It is important to appreciate that *not* all pericardial effusions produce electrical alternans. Development of ***total electrical alternans*** (*of P wave, QRS complex and T wave*) — is likely to be a harbinger of ***impending tamponade***. Unfortunately — the sensitivity of *total* electrical alternans is poor for predicting tamponade (*ie, most patients who develop tamponade do not manifest preceding electrical alternans*). Therefore — it may

be helpful if you see total electrical alternans in a patient with a large pericardial effusion — but failure to see this ECG sign *in no way* rules out the possibility that tamponade is occurring. Echo studies in patients with documented cardiac tamponade confirm that electrical alternans is synchronous with and a direct result of the *pendulous* movement of the heart within the enlarged, *fluid-filled* pericardial sac of a patient with large pericardial effusion (*See Section 14.4*).

14.2 – Electrical Alternans: **KEY Clinical Points**

In summary, electrical alternans is *not* common — but it does occur. You will see it. You have probably *already* seen it a number of times without even realizing it. **Electrical alternans** is a fascinating but advanced concept. It is clearly beyond-the-core for many who are using this ECG-2014-ePub — but we choose to include it because of its uniqueness and the clinical insights that this fascinating ECG sign may provide.

- In our experience — **electrical alternans** is most often seen in association with **regular SVT rhythms**. Seeing it in this context suggests (*but does not prove*) the existence of an AP (Accessory Pathway). Regardless of whether the mechanism of the *regular* SVT is AVNRT or AVRT — it is likely that **reentry** is involved. This conclusion may prove useful in contemplating potential investigative and therapeutic interventions.
- In a patient with pericarditis — a large heart on chest X-ray — or simply unexplained dyspnea — recognition of electrical alternans should suggest the possibility of a significant **pericardial effusion** that may be associated with tamponade. That said — electrical alternans is a **nonspecific ECG sign** that may also indicate myocardial ischemia, LV dysfunction *and/or* possibility of any of a number of *other* precipitating factors. **BOTTOM Line:** If you see electrical alternans — Look for an *underlying* clinical condition that may be responsible for this ECG sign.
- Development of **electrical alternans** per se — conveys *no* adverse prognostic implications *beyond those* associated with severity of the *underlying* disorder. Two exceptions to this general rule are: **i)** In a patient with QT prolongation or severe ischemia — recognition of electrical alternans may portend *deterioration* to Torsades or VT/VFib; and **ii)** In a patient with a large pericardial effusion — development of **total electrical alternans** (*of P wave, QRS complex and T wave*) suggests there may now be tamponade.

14.3 – FIGURE 14.3-1: **Alternans in an SVT Rhythm?**

Consider the 3-lead rhythm strip shown in Figure 14.3-1 — obtained from a patient in a **regular SVT rhythm**.

- Is electrical alternans present? If so — *What kind* (ie, *involving the P wave; QRS complex; ST or T wave; or involving the PR or R-R interval?*).
- What are clinical implications for electrical alternans of this rhythm?

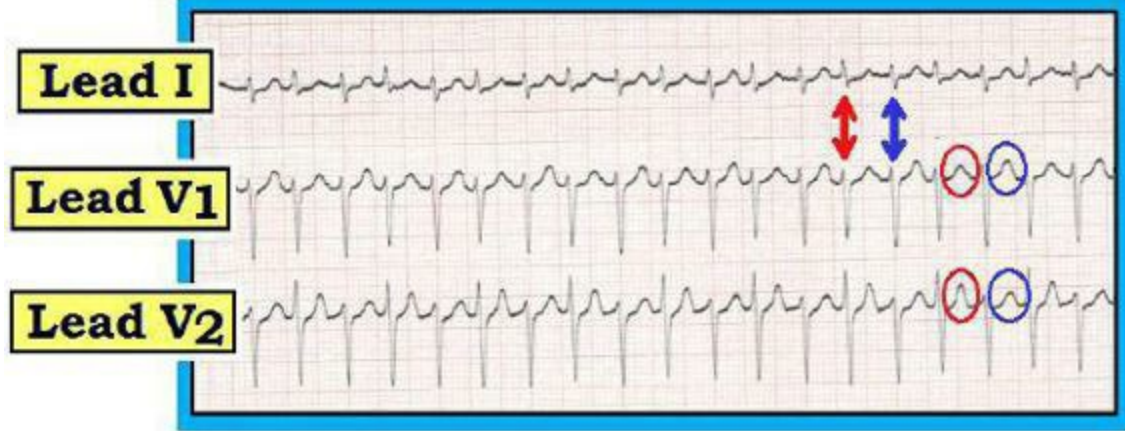


Figure 14.3-1: 3-lead rhythm strip — obtained from a patient in a *regular* SVT rhythm. Is electrical alternans present? If so — *What* kind? (*See text*).

Answer to Figure 14.3-1: Although we describe the rhythm seen here as a “*regular*” SVT (*SupraVentricular Tachycardia*) — there appears to be *slight-but-real* phasic variation in the R-R interval occurring every-other-beat (*ergo*, **R-R alternans**). In addition — there is both **QRS alternans** (*red and blue double arrows in Figure 14.3-1*) — and **T wave alternans** (*red and blue circles*). That is — QRS morphology *changes* every-other-beat. This is subtle in lead V1 — but more noticeable in lead V2 where the initial R wave manifests an obvious difference in height from one beat to the next. Similarly — T wave morphology changes every-other beat, with this clearly more noticeable in lead V2 which manifests extra peaking of every-other-T wave (*red and blue circles in lead V2*).

- Clinical implications of these forms of electrical alternans in a patient with SVT — are that **reentry** is almost certain to be involved in the mechanism. There may or may not be a concealed accessory pathway.

14.4 – FIGURE 14.4-1: *Alternans in a Patient with Lung Cancer?*

Consider the 12-lead ECG and accompanying rhythm strip in Figure 14.4-1 — obtained from a longtime smoker who presented with *new-onset* dyspnea. Pulmonary nodules suspicious of cancer had been identified on this patient’s admission chest X-ray. In addition — a *large* heart shadow was seen.

- Note the *bigeminal* pattern for the ECG in Figure 14.4-1. Is the rhythm ventricular bigeminy (*every-other-beat a PVC*)?
- Given the history — What clinical entity should be considered?
- What might this patient’s Echocardiogram show?

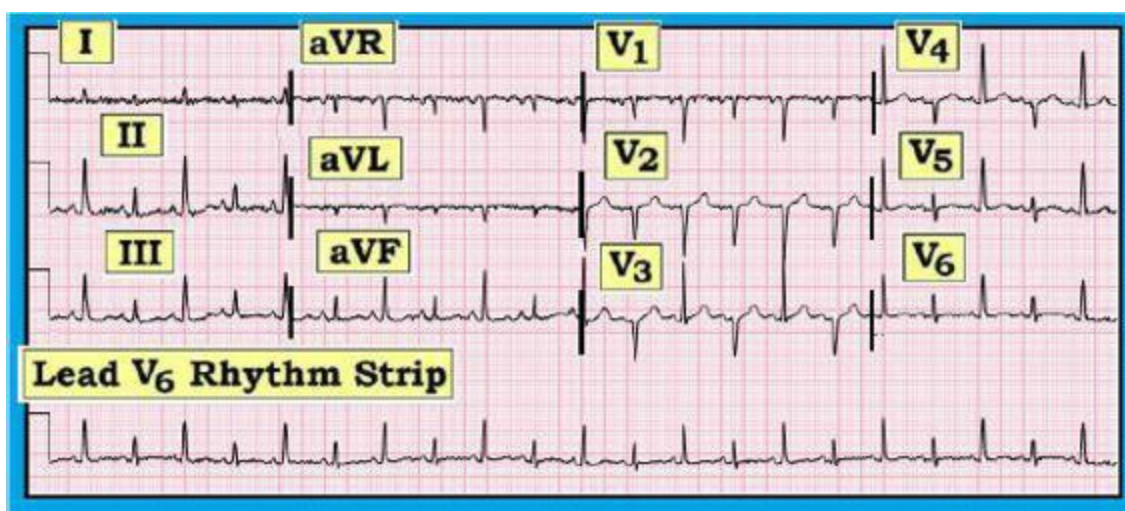


Figure 14.4-1: 12-lead ECG and lead V6 rhythm strip — obtained from a patient with dyspnea and suspected lung cancer. What is the *likely* cause of the *bigeminal* rhythm? (See text).

Answer to Figure 14.4-1: There is obvious **QRS alternans** — with marked variation in QRS amplitude from beat-to-beat. This is most dramatic in lead V3 — in which there is a 180 degree alternation in QRS direction from one beat to the next.

- The rhythm is *not* atrial or ventricular bigeminy — because P wave morphology is constant and the P-P interval is perfectly *regular* throughout the tracing. The PR interval remains the same. Therefore — the rhythm is sinus and the change in QRS morphology must be solely the result of **electrical alternans**.
- ST-T wave morphology does *not* appreciably change from beat-to-beat in any of the 12 leads on this tracing. Thus, alternans appears limited to a change in QRS morphology.
- **Clinical Note:** Knowing this patient's history supports our presumption of electrical alternans. The patient is a longtime smoker suspected of having lung cancer. He now presents with acute dyspnea and a large heart shadow on chest X-ray. In view of **electrical alternans** on ECG — a large **pericardial effusion** with possible tamponade should be suspected. An **Echo** should be done in timely fashion (**Figure 14.4-2**).
- **Beyond-the-Core:** Overall QRS amplitude appears *reduced* in **Figure 14.4-1** — especially for *every-other-QRS* complex. The finding of **low voltage** in the context of the above history and electrical alternans all support the likelihood of finding a significant pericardial effusion. An **Echo** is the investigative procedure of choice (**Figure 14.4-2**).



Figure 14.4-2: Serial 4-chamber views from the Echo performed on the patient whose ECG was shown in **Figure 14.4-1**. An extremely large pericardial effusion is seen (*arrows*). As a result — a

“*swinging heart*” pendular motion is set up. This marked *free-floating* displacement of the heart occurs in phasic fashion within the *fluid-filled* pericardial sac — and accounts for the dramatic *beat-to-beat* variability in QRS amplitude and direction that was seen in Figure 14.4-1.

ACKNOWLEDGMENT: My appreciation to Jenda Enis Stros for allowing me to use the ECG in Figure 14.3-1 — and to Jason Roediger for allowing me to use the case, ECG and Echo in Figures 14.4-1 and 14.4-2.